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# **BORDERLINE DISEASES**



# BORDERLINE DISEASES

A STUDY OF MEDICAL DIAGNOSIS WITH  
ESPECIAL REFERENCE TO ITS SURGICAL  
BEARINGS

BY

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PEUTIC SOCIETY, THE AMERICAN ACADEMY  
OF MEDICINE, ETC.

*WITH ONE HUNDRED AND FIFTY ILLUSTRATIONS  
IN TEXT AND ONE PLATE*



VOLUME I

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## PREFACE

THE especial aim of this treatise beyond the usual objects of a work upon medical diagnosis is the particular study of certain conditions in which accurate diagnosis is essential to the proper decision as to the advisability of surgical intervention.

In the consideration of the diseases of the abdomen, we note the most frequent and urgent need of good team work by the physician and the surgeon, but in other departments of medicine a similar demand exists. The fault has lain as frequently at the door of the physician as at that of the surgeon for the occasional useless operations for removal of the appendix when the true diagnosis was acute pneumonia; the performance of gastro-enterostomy in the gastric crises of tabes, or the surgical drainage of the bladder for supposed cystitis when the true diagnosis was that of tuberculosis of the kidney. Such humiliating experiences are now commonly avoided by better and more systematic coöperation between the diagnostician and the surgeon.

The attainment of such exact results as may be obtained by a Cabot in hematology or a Mills in cerebral localization is beyond the possibilities of abdominal diagnosis. In the more urgent instances the really important question—that of deciding upon the advisability, or, oftentimes, the imperative necessity of exploration—is capable of quite exact decision although the precise diagnosis be not always within our reach.

On the other hand, a host of invalids travel from one physician or one sanatorium to another, labelled with the diagnoses of indigestion, chronic dyspepsia, nervous dyspepsia, gastralgia, biliousness, etc., in whose cases the diagnosis of chronic appendicitis, gall-stones, peptic ulcer, intermittent hydronephrosis, or other definite disease is perfectly possible. The diagnosis should be made and should be followed in many cases by that absolute cure which is possible and only possible by surgical intervention. I venture to hope that in this particular field this embodiment of my experience may possibly be of assistance to some members of the profession, for not all have been so fortunate as I have been in my close association with a group of able surgeons. To them I owe the major part of whatever may be of value in this work.



It is a difficult and costly process to reproduce röntgenograms in full detail in ordinary book-making; those presented in this book have been selected with a view to giving a reasonable illustration of the text matter, but recognizing the fact that all the detail of a plate cannot be brought out by half-tone cuts on book paper.

I wish to express my thanks to Dr. J. W. Ames for assistance in the subjects of Tropical Diseases and Diseases of Children; and to Drs. M. Black, Robert Levy, and C. L. Pershing for assistance in the sections dealing respectively with the Eye, the Nose and Throat, and the Nervous System; to Dr. G. H. Stover and Dr. S. B. Childs for their excellent röntgenograms; to Dr. Leonard Freeman for invaluable assistance upon surgical subjects; to Dr. J. R. Broome of D. Appleton and Co., for numberless courtesies; and above all to Dr. T. R. Love for his especial assistance in compiling my records and preparing many of the illustrations.

J. N. HALL.

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## INTRODUCTION

By diagnosis we mean the classification of a given disease under its proper heading. Inasmuch as its object is to enable us to forecast the probable course of the disease and to treat it in the best manner, we should include in our diagnosis as close an estimate as possible of the severity of the affection, whether organic damage is already present, and whether it is capable of entire restitution. The diagnosis is incomplete if it does not give us a fairly complete grasp of the patient's condition and prospects.

Diagnosis is often impossible for the reason that the signs and symptoms in the given case have not developed far enough to furnish a sufficient basis for a diagnosis. This is no reproach to our art, for a similar condition is found in many other fields of knowledge. The botanist cannot tell from the first green shoot whether he is dealing with a sprouting grain of wheat or rye, but the diagnosis is easy later. Most diagnoses of important diseases may be made with reasonable accuracy and promptness provided only that the indications of the affection are sufficiently manifest.

The history may be wanting, the physical signs may be impossible of detection, the patient may have a rare or even a new disease, or may have two or three diseases associated together. We cannot reason correctly without all the material facts, and it is impossible to obtain them all in many cases. Yet practical diagnosis is more certain than we should theoretically have supposed would be possible.

A diagnosis is based upon subjective symptoms, which are manifestations of the action of some morbid process perceptible to the patient, and objective symptoms, signs or physical signs, as they are variously called, determined by the physical examination made by the physician. Occupation, age, hereditary tendencies, habits, previous illnesses or injuries, temperament and place or places of residence must be considered.



To the facts gathered by these processes of observation it is next essential to apply a process of reasoning. Though all the facts be correctly ascertained, without correct reasoning we shall fail in diagnosis, while conversely, the best reasoning may be of no avail if material facts escape us. Many rather unusual diseases will pass unrecognized if we are not alert in thinking of the relationship between occupations, places of residence, personal experiences, matters of family history, etc., and the possible diagnosis. All of the peculiarities of the clinical history will not be brought out in the usual anamnesis and we must have a ready ear for anything mentioned which may bear upon the case, unless it be a very ordinary one, to make a correct diagnosis. The Japanese patient may have hemoptysis and we think first of pulmonary tuberculosis, but the bronchial fluke is the common cause of pulmonary hemorrhage in Japan. An army officer with multiple neuritis may be known to use alcohol to excess, but he may have served in the East and suffer from beri-beri, he may have had some acute infectious disease, may have been using arsenic for chronic malaria, etc. The possible bearing of anything mentioned must be quickly appreciated.

The art of diagnosis consists, then, in gathering all accessible data and arriving at that conclusion which seems most reasonable as the probable cause of the trouble. No mathematical accuracy can be expected. The factors upon which diagnosis is based are variable ones in different cases and never have exactly the same weight. Inasmuch as some of the most important ones may be absent, the reason for possible error in diagnosis may be more readily appreciated. In no department of human endeavor does the element of sound judgment enter to a greater extent than in medical diagnosis. One may be a great mathematician without this endowment; two and two always make four, but in medicine we are called upon to judge as to the value of variable factors. Unless one can pick out a discordant falsification in a given history, and estimate the value of each sign and symptom with fair accuracy in the given case, he cannot work out the right answer to the clinical question.

A certain finding may be pathognomonic, indicating with certainty that a given disease is present, as, for example, the detection

of the malarial parasite in the blood. It may be practically pathognomonic, as in the case of thoroughly characteristic rusty sputum in pneumonia, or a typical rash in measles, requiring only other reasonably confirmatory evidence. The symptom may be of great weight, but pointing only in a general way toward either of several diagnoses, the exact one being found only by means of associated signs and symptoms. Thus severe colicky pain in the upper abdomen may be due to gall-stone disease, gastric ulcer, tabes, etc. If jaundice, hematemesis, or ataxia be present the pain is equally valuable, but as a factor in a different diagnosis in each case. Then the symptoms may be of value only in such a general way as to indicate that the patient is ill, without any specific indications as to the diagnosis. Fever, emaciation, and debility may be mentioned under this heading.

Then other symptoms are of practically no value in serious cases because so common that their presence or absence would not influence us. Constipation of moderate degree is an example.

The failure to mention a certain symptom or the denial of its presence when questioned may be a matter of intentional deception, or of ignorance or inattention. The denial of a previous urethritis when gonorrheal threads appear characteristically in the urine should be placed under the first heading, a specific eruption may be denied solely through ignorance, while a pigeon breast may be stated to have been present but a few days solely through inattention.

A diagnosis may be placed under any of the following headings: anatomical—based upon a post-mortem examination or upon definite anatomical changes discovered in the examination, or more especially in operation; pathological—relating to the pathological changes present in parts affected by the disease; clinical—based upon symptomatic evidence only; presumptive—sufficiently clear to serve as a basis for treatment, but conveying prominently the idea that further evidence may invalidate it.

Differential diagnosis is a term applied to the process of reasoning whereby we determine between different affections presenting somewhat similar characteristics. If we finally exclude all but one disease, we have arrived at a diagnosis by exclusion. A designation

for which there is abundant warrant is that of a "working diagnosis." I should define it as such an approximate diagnosis as suffices for laying out a proper course of treatment, more especially surgical, in a given case, without an attempt to define with great accuracy the exact location or nature of the lesion, excepting in broad terms. Thus a great number of patients have digestive disturbances with tenderness, rigidity, and pain in the region of the pylorus and gall-bladder. If such patients fail to improve under medical treatment, but on the contrary, show more and more evidence that a serious and incapacitating illness exists, the abdomen should be explored. All the usual means should be followed to arrive at as exact a diagnosis as possible, but when doubt remains, as is so often the case in this particular field, the exploration should follow. In this case we may be reasonably certain that peptic ulcer, pyloric cancer, gall-bladder disease, or some such serious condition exists, but the digestive apparatus in this particular region is so delicate, and each part so intimately related anatomically and functionally to the neighboring ones, that exact diagnosis is frequently impossible, though comparatively easy upon opening the abdomen. In the case of a perforation of the stomach, bowel, appendix, gall-bladder, etc., the points to be considered are chiefly the location of the lesion, as a guide to the incision, and the probable ability of the patient to survive the operation long enough to justify it. Time here is so overwhelmingly important as to preclude tedious history-taking or examination.

My own case histories have for many years been kept with reasonable accuracy, but with utmost brevity, upon cards three by five inches in size, indexed according to the diagnosis and cross-indexed according to the name. The odd minutes of the day fairly suffice to keep them up since they are carried in an ordinary prescription book and may be completed while one waits for a train to start, a colleague to appear, or an operation to begin. Additional notes are made upon prescription blanks, etc., to be copied upon the proper cards by the one in charge of the indexing. It is much more satisfactory and accurate to note the operative conditions before leaving the operating room. Where more than one disease

worthy of regard is found, the second one is listed upon the guide or index card of that disease. Thus if the main trouble is ulcer of the stomach, but gall-stones be also found upon operation, the patient remains listed under the first heading, but upon the guide card headed "gall-stones" appears the note, "See John Smith, under Stomach, Ulcer of." In this way all case histories of gall-stone disease are easily gathered together if needed. Separate cards for urinalysis, temperature, and pulse, blood examinations, etc., may be used if desired, but I have generally made such brief notes as seem to be desirable directly upon the history card. Outline stamps are often of value. After trying various methods of printing cards I have tried to reduce the whole matter to its simplest terms by writing what was essential upon the blank card. Elaborate and cumbersome records cannot be kept by one who becomes embarrassed with his professional work and therefore such a simple plan should be adopted early in life as will not be burdensome later.

## SAMPLE HISTORY CARD

Heart Disease.	John Smith—28—S
(mitral.)	Blacksmith. Fort Lupton.
March 1, 1909, with Dr. L. J. Jones.	
Acute rheumatism at 17 and at 23—severe in each case. Heart affected in first attack. Had dyspnea, and frequent attacks of palpitation following it. Has recently become unable to work on account of shortness of breath, swelling of feet and general weakness.	
(Description of cardiac condition).—Urine—trace of albumin—no sugar—moderate number of hyaline and granular casts.	

Further notes may be placed on the reverse side of the card. Two or more cards may be clipped together if desirable.

## SAMPLE INDEX CARD

John Smith.	Heart Disease.
March 1, 1909.	(mitral.)

The History or Anamnesis.—After noting the name, age, sex, occupation, civil condition (single or otherwise), and the residence of the patient, with the date of examination, we ask of him if there is any especial family tendency to disease. One may ask, "Has there been any particular disease especially common in your family?" Next we may ask if the patient has ever been seriously ill. The symptoms may now be noted, but one should seek for certain evidence in every case, e.g., as to cough, digestion, passage of urine, movement of the bowels, habits as to alcohol, coffee, etc., menstruation in women, miscarriages, etc. If the history be given in such a rambling, aimless, time-consuming way as to make it necessary, one may adopt the plan of asking answers to direct questions. In such a case opportunity should be given later for the mention by the patient of anything not properly brought out. It is not necessary to ask in every case as to the presence of venereal disease, hereditary syphilis, pregnancy, alcoholism, etc., but the physician should be alert to perceive the slightest evidence that any such condition exists, and to follow it up as required. Nor should one's ideas of the clinical manifestations of the disease be influenced in the slight-

est degree because of the age, occupation, social standing, or supposed moral standing of the patient. When the physician declines to ask for the history of gonorrhea in certain bladder diseases, of syphilis when a double tibial periostitis exists, concerning the use of alcohol when a well-dressed woman complains of numbness and pain in her fingertips, or the use of snuff or cigarettes when attacks of otherwise inexplicable palpitation exist, or of pregnancy in a young woman with morning vomiting and suppressed menstruation, he jeopardizes at once his patient's welfare and his own reputation. The inquiries should of course lead along the line of the symptoms, in many of these instances, but where suspicion points with sufficient force we must put the burden where it belongs by direct questions. In five instances out of some two hundred cases of functional cardiac disease I found the trouble due to use of cigarettes in two women, to using snuff in two, to chewing tobacco in one. A lack of this knowledge would have defeated every effort toward cure in each of these cases.





**PART I**  
**GENERAL CONSIDERATIONS**



## SECTION I

### EXAMINATION OF THE PATIENT

THE history of the case having been ascertained we should proceed with the general examination of the patient, this to be followed by special examination as the developments of the case seem to require.

The dress, general behavior, condition of nutrition, color, posture, gait, intelligence and general character of the patient are noted almost unconsciously, if one be sufficiently familiar with mankind in general. The young physician should take every opportunity to see disease in or otherwise come in contact with, people of all social conditions and of different races, in order to broaden his foundations. Many of the diseases rarely seen in this country have come under my own observation in those foreigners from almost every land whom one may find in any large city. A little time spent with a Japanese physician, for instance, in examining one of his countrymen, may bring small remuneration, but as tending to exercise one's powers of observation, to cultivate one's tact, to enlarge one's field of clinical experience, such time is well spent.

When certain obvious features like the high fever and red sputum of the patient with acute pneumonia lead the physician at once to the seat of the trouble, a systematic examination into the history of the patient for the purpose of diagnosis is not imperative. In many cases where the symptoms are more or less indefinite, however, a regular plan should be followed. This must be sufficiently comprehensive to include every important element necessary to the detection of notable disease in any set of organs in the body. Yet, if too prolix, it either consumes more time than it is possible to devote to the average case or is so habitually slighted as to be of little value.

There are many diseases indicated by some particular sign or symptom which are perfectly easy of diagnosis if we have our attention called to the possibility of their occurrence, yet are easily

overlooked or forgotten if not so sought for. Many times we may overlook the key to the whole case by simple omission, if not accustomed to look in every place where important evidence is to be found. In the abstract of title to real estate we may see the question, "Was the notary's seal attached?" This question is no more easily answered than that of whether a lead line was present on the gums, but as easily neglected if not specifically looked into. Our system should guard us against omitting any of the prime factors in diagnosis, and must, therefore, include an examination into many points where our results will be successively negative in scores of cases. Yet the positive result in a single case justifies us in examining all. I do not see the lead line mentioned more than twice a year, perhaps; yet its value is so striking in the few cases where it is found, and the suggestion of metallic poisoning, that the very act of seeking for it involves, is so valuable, that I consider no examination complete unless it is included.

I have long been accustomed to follow the routine given below in most cases, excepting in those of rather trivial and quite apparent nature. The taking of the usual history will show one enough of the patient's mental condition for a proper judgment as to probable mental disease. The pulse and temperature are noted before the regular routine is carried out in most instances.

Characteristic attitudes should be noted. In certain cases of chronic appendicitis or, more especially, cases in which an ascending infection from suppurative appendicitis has caused a subphrenic abscess or abscess of the lung, and occasionally in duodenal ulcer, the patient bends to the right. A tumor of the abdomen or pregnancy causes a backward bending to bring the center of gravity in the proper place, while a large fatty tumor of the back of the neck causes an opposite position to be assumed. Sciatica of long standing causes the patient to bend toward the affected side, rarely in the opposite direction. Paralysis agitans leads to the "falling-forward" position. An inflamed hernia causes a sharp anterior bending at the groin, as does a bubo. The attitudes assumed in congenital dislocation of the hip, ankylosed hip joint, in flat-foot, in lumbago, goiter, blindness and deafness need no description. The patient with

herpes zoster often leans toward the affected side, to keep the clothing from chafing the hyperesthetic skin, so that his trouble may be suspected from this position alone. The painful pustules arising from the application of croton oil to the front of the chest cause a forward bending of the upper part of the body for a similar reason. The assumption of a peculiar attitude in coughing may indicate that the patient has learned from experience that his expectoration is easier because of this procedure. A unilateral cavity or bronchiectasis may be suspected. In a similar way, suppuration of the antrum of Highmore causes certain patients to tip the head forward when blowing the nose, with the face so placed that the infected antrum is higher, since in this position it more readily empties itself into the nasal passages.

One should always look carefully for the signs of paralysis of the face, since the first intimation of hemiplegia is often seen there, and, in an unconscious person the hemiplegia is extremely likely to be overlooked. It is especially important to look for the signs of a slight paresis, often the forerunner of a complete paralysis, which may be the first definite evidence of meningeal inflammation or pressure, thrombosis or other cerebral condition.

A close scrutiny of the face (*see* the Section on Examination of the Face) should be followed by a careful look at the eyes. One can determine, in a fraction of a minute, if they have parallel axes, if the pupils are of equal size, if they react to light, if they move in proper coördination, and whether or not arcus senilis, nystagmus, or exophthalmos be present.

If any head symptoms have been mentioned, the ears should be examined tentatively as to the sense of hearing by asking if the patient hears the physician's voice more distinctly in either ear when the other one is closed. Discharge from the meatus, tenderness upon movement of the external ear, or from pressure upon the mastoid, is easily determined. Enlargement of the glands in the neck may be significant. The nose, lips, gums, teeth, tongue, palate and pharynx are next noted. The size of the thyroid gland, and, in case intrathoracic disease is suspected the presence or otherwise of tracheal tug are observed.

After stripping the chest one should look carefully in good light to determine its shape and mobility. The position of the apex-beat, the presence of abnormal pulsation, limited respiratory movement or of enlarged veins, glands, tumor growths, etc., should be noted. The percussion of the chest, if no local disease be suspected, may be briefly carried out and yet guard one against overlooking lung disease, or silent pleural effusion, aneurism, mediastinal growth, pneumothorax, dilated heart, hydropericardium, transposition of the viscera, etc. If anything abnormal develops, a further examination is demanded. If this ordeal be safely passed the auscultation may be similarly brief. If the apex-beat be in normal position and the heart sounds of proper force and character the heart may be passed for the time. It takes no longer to feel both radial pulses than one, and the finding of deficiency or retardation upon one side would lead at once to a fuller investigation. The condition of the arteries is noted at this time.

For the examination of the abdomen the patient should be placed flat upon the back in good light, preferably coming from the foot of the table. Any tumors or glands projecting from the abdominal wall should be noted, for they may indicate malignant metastases, small omental herniæ, etc. Enlarged veins and the direction of their current deserve especial attention, since they often serve to show that a blocking exists in either the superior or inferior vena cava, or in the portal system. Jaundice is seen at the navel before it shows elsewhere in certain cases. The shape and mobility of the abdominal walls should be carefully observed. Bulging in different regions may suggest very strongly different diseases. If the lower central part of the abdomen be protuberant in a female, pregnancy should first be thought of, or a distended bladder in either sex. If the lateral walls bulge, while the center is flattened, effusion is likely to be found. Uniform distention is generally due to tympanites or extreme ascites. Bulging in the right upper quadrant suggests cancer or other tumor of the liver, subphrenic abscess, or, in a child, sarcoma of the right kidney. If the right side projects very sharply and jaundice be present, Hanot's cirrhosis must be considered. If the upper left quadrant is enlarged, close up to the tip of the ster-

num, pancreatic cyst is suggested. If the ribs are pressed outward and the waist increased in diameter, splenic leukemia is often present. In the lower right quadrant we see the tumor of appendical disease, often accompanied by respiratory immobility. The left quadrant is relatively less often changed than the other three, sigmoidal disease and pelvic abscess or pelvic tumor being the chief features. A floating kidney may distend the lower right quadrant, in very thin persons, and occasionally the left. Malignant disease in any given locality may cause projection of the abdominal wall, the most common seats in the abdomen being over the liver and stomach regions.

Next we should percuss out quickly but sharply the liver and splenic areas. If the former procedure be carried out with any care, transposition of the viscera will infallibly be detected. Though this condition is rare and generally of no special importance, I cannot conceive a greater shock to one's own estimate of his acumen as a diagnostician than to learn that he has overlooked it. I have found five cases in fifteen years, two of them accompanied by congenital heart disease, so that we may conclude that it is not a condition of extreme rarity.

The area of the stomachic resonance is generally determined with little difficulty by ordinary mediate percussion, or by immediate percussion performed while the bell of the stethoscope is held over the center of the area of resonance. If doubt exists the distention by air or gas is easily performed. One can readily hear the "sizzling" caused by the ingredients of a Seidlitz powder uniting in the stomach—decisive as to its position even if the general shape of the organ or the gurgling produced by the gas is not conclusive. Dullness in the center above the pubes suggests in order, a full bladder, pregnancy, or a pelvic abscess or growth. If the abdomen be resonant in the center and flat at the periphery, free fluid is probably present. Large appendical and pelvic abscesses are often to be located by percussion. Extensive dullness over the spleen suggests leukemia, splenic anemia, etc., while the large area of liver dullness brings to mind abscess, cancer, hypertrophic cirrhosis, hydatid disease, etc.



We should now examine for tenderness and rigidity, and with especial care, since many troubles which have not yet revealed their presence may now be found. The regions of the gall-bladder, appendix and pylorus, and the center of the epigastrium must be examined closely. If any tenderness be found, rigidity will probably be present, and it is even more significant, as being less subject to modification by the patient's will, and less likely to be overestimated because of his complaint.

The recti muscles should be compared with extreme care. In case the right one be more rigid, appendical disease, gall-bladder trouble, duodenal ulcer, pyloric ulcer or cancer, movable and tender right kidney, cancer and other disease of the liver, pelvic abscess, tubal disease, and tuberculosis of the caecal region pass before the mind. If no rigidity is found here, we should try the region between the crest of the ilium and the ribs, where rigidity is often noted in appendicitis, when the affected organ is placed behind the cæcum. Upon the left side rigidity is likely to indicate ulcer of the stomach elsewhere than at the pylorus, pancreatic, splenic or sigmoid disease, or the pelvic troubles mentioned, upon this side. Too much stress cannot be laid upon this sign, nor upon the need of the utmost care in examining for it. If the rigidity be general and well-marked, excepting in the occasional neurotic or ticklish individuals, a general peritoneal involvement is to be sought for.

The size, consistency, shape, position and mobility of the liver, spleen, kidneys or other abdominal organs or growths are to be investigated. In patients with any signs or symptoms arising from the region below the navel, or with backache in or below the dorsal vertebræ, a rectal examination should be made, or in women a vaginal one. A simple digital examination will tell us most of what we need to know. In the male, hemorrhoids, fissure, cancer of the rectum, appendical abscess pointing downward, prostatic disease and seminal vesiculitis will be detected almost instantly, and any one of these might be easily overlooked without this examination. In women the usual pelvic diseases and pregnancy must be thought of.

A brief palpation of the axillæ and groins will show any glandular involvement. If any digestive disturbances be present it is

imperative to examine the hernial openings, including the navel, and to palpate the central line of the abdomen, especially above the navel. An omental hernia of the size of a grain of wheat in the central line may cause more troublesome symptoms than a large gastric ulcer, as I have often observed.

Edema of the feet should be sought for regardless of the patient's statement as to its presence or absence.

If there have been any symptoms pointing toward the pelvis or the lower limbs, the patient, if physically able, should be made to rise upon the toes and descend sharply upon the heels. If bony disease of the spine or pelvis exists, it is likely to be determined at once by the pain caused by this procedure. The rather common osteoarthritis of the spine, tuberculous disease of the spine or pelvis, loosened sacro-iliac articulations, as after confinement, typhoid spine, etc., are to be looked for. If this procedure be without pain, antero-posterior and lateral movements of the spine are to be tested. A beginning osteoarthritis may limit motion in only one direction, and but slightly, and yet make an invalid out of an otherwise strong man.

The knee-jerks and station of the patient may now be tested, and the gait if necessary. If any pains in the feet and legs be not otherwise easily accounted for, the arch of the foot should be inspected while carrying the patient's weight. Inversion of the ankle and flattening of the arch are so commonly the cause of so-called "rheumatic pains" that they must not be overlooked.

The urine, sputum, blood and stools are examined as may be necessary, although the urine should rarely be passed over even in apparently trivial cases.

Through such an examination as we have described, we should be able to find sufficient evidence of any disease present to point us in the direction of the proper diagnosis.

Serious mental diseases will generally have been detected in the taking of the history, or else suspected because of the unequal pupils, tremor about the lips and tongue, changed knee-jerks, etc. The proper coördination of the eye muscles, reaction to light and accommodation, absence of tremor in face and hands, presence of normal knee-jerks, absence of tenderness about the ear and mastoid

process, absence of Romberg's phenomenon, absence of signs of paralysis, ability to rise on the toes in testing the integrity of the spine, and the history of the normal action of the bowels and bladder, throw out at once most of the ordinary organic nervous diseases. No serious disease accompanied by physical signs should escape us in such an examination of the chest, abdomen and pelvis. The absence of the lead line and of paralysis of the extensors should prevent our overlooking one of the most elusive diseases, lead poisoning. The normal pulse in both wrists, equal pupils and the absence of abnormal dullness or pulsation in the chest, and of the tracheal signs, with a normal voice, will prevent our humiliation at the non-discovery of aortic aneurism, so commonly overlooked. Leukemia and other blood diseases will be suspected through the results of the examination of the spleen, liver, and glands, and confirmed by an examination of the blood. The examination of the urine as to its color, clearness, presence or absence of blood, pus or bile, and of albumin, sugar or sediment, will show us any trouble connected with the urinary organs. If any bowel trouble is unaccounted for, the examination of the stools will usually make it clear.

Certain diseases, like asthma, hay fever, epilepsy, recurring appendicitis, gall-stone colic, etc., will not be revealed unless they cause some disturbance at the instant of the examination, but a properly given history should save us from error. It will be readily understood that much of the value of such an examination lies in the fact that, if properly carried out, it brings before the mind certain possibilities of disease. Thus the very act of feeling both radial pulses will bring the possibility of aneurism so sharply before one's mental vision as to prevent its being overlooked if any sufficient sign exists for its diagnosis.

### THE HEAD AND FACE

After taking such a history as I have indicated, the size and shape of the head are noted, and the condition of the fontanelles, especially if they be too large. In this case we should not neglect to percuss the lateral regions of the head directly with the tip of

the finger. The tympanitic note often obtained is significant of the presence of fluid in the lateral ventricles. Difference in resonance upon the two sides often appears with intracranial growths, and abscess and tumor often present acute tenderness over their area of contact with the skull.

The face is scrutinized as to its color, nutrition, expression, scars, deformities, paralysis, edema, eruptions or other features. All physicians, in a way, go through this formula, but some fail to gather all that may be learned, chiefly from inattention to detail.

The facial expression may be most suggestive, as in the dull, stupid look of typhoid fever, the lethargic face of myxedema, the inexpressive one of paralysis agitans, the frightened expression of exophthalmic goiter, the distressed one of painful abdominal disease, the "suffocative" one of obstructed aëration of blood, the enlarged and cyanotic one of advanced cardiac disease, the excited one of acute mania, the sad one of melancholia, the ecstatic look in trance, the confident, swaggering expression in the expansive stage of general paralysis, and the various hysterical conditions. The facial expression may at times almost serve to differentiate certain diseases. The decided expression of apathy and resignation in the patient with internal cancer contrasts sharply with the generally alert and interested look of the one with gastric ulcer. Pernicious anemia resembles cancer in this regard.

Much may be told by scars on the face; the usual ones of small-pox whether confluent or discreet, need only be mentioned. Perhaps half our patients have a round scar or two from chicken-pox. The scars from pustular syphilid are finer and often closer together than those of small-pox, while those of acne are often coarser. The extensive scars of lupus are less common here than in large European clinics. The scars from accidental wounds are often of little consequence, but may suggest occupational diseases. The bluish-black irregular cicatrices, perhaps up to a dozen in number, often seen in miners, are the result of an explosion, and are due either to pieces of coal or dark-colored ore driven into the face, or to the coarse, black blasting powder still used in coal mining. Marks from the latter cause are more uniform in size than those from coal or ore. The

stronger nitro-powders leave less stain but much more frequently destroy the sight. In regions where much rock work is done the miner with the sight of one or both eyes and parts of both hands missing, and the face filled with irregular scars is a common sight—the effect of an untimely explosion of a charge of dynamite-containing explosive which has blown up in the attempt to remove the missed shot. If such a patient coughs, the suggestion of miner's phthisis is very strong. The common scars from fireworks and from accidents with firearms are characteristic, but of comparatively little diagnostic value.

Terrible scars result from the free openings necessary in the treatment of phlegmonous erysipelas. The scars from burns and scalds are noted with the resulting contractions. Frequently a fairly right-angled dark discoloration is seen when a mustard plaster has been allowed to remain too long over an inflamed antrum, or facial neuralgia. In negroes, scars upon the face, especially below the angle of the jaw, often show the marked hypertrophy of Alibert's keloid.

Depressed adherent scars in the cheeks often result from the perforation of abscesses about the teeth and from gunshot wounds. Fistulae, from which saliva escapes, indicate involvement of the salivary duct. Irregular scars in the lateral region of the neck bespeak the natural openings of tuberculous abscesses, those opened by the surgeon showing less deformity. Over the parotid gland may be seen the scars from opening of suppurative parotitis, generally indicative of typhoid fever or other serious febrile disease. The puckered scar of natural evacuation may be seen, externally, or in the anterior wall of the auditory canal. Rarely the enlargement at the angle of the jaw due to branchial cyst may be noted. (*See Fig. 1.*)

A purulent nasal discharge accompanied by pustules within the nares should suggest glanders. It is common for the irritating discharge from an inflamed accessory sinus to cause the opening of the nostril upon that side to show redness and excoriation. The increased discharge from one nostril mentioned as occurring in facial neuralgia, is doubtless often better explained upon the grounds just given.

The tip of the nose is lost from freezing at times, and in Russia, from spontaneous gangrene. Greater deformity results from loss

by a bite, not infrequent in drunken brawls. The sunken bridge of syphilitic disease may be closely imitated by traumatic cause as the kick of a horse, but the history makes the case clear.

Inequality of the pupils should be especially noted. It may give the first hint that abdominal pain is of tabetic origin (tabetic crisis), since it is extremely often of syphilitic origin, and syphilis lies at the basis of most cases of locomotor ataxia. It may be the first sign of brain tumor, cerebral thrombosis, or other vascular disease, syphilis of the brain, etc. Its frequent presence after injury to the eye must not be overlooked, while it is of no diagnostic importance in certain other cases. Irregularity of the pupil may be noted in the above conditions, and it is also seen after iritis of rheumatic, syphilitic or other origin. If one finds the pupils sharply dilated at intervals, upon different examinations of the patient, the possibility of the use of cocain should be considered. If the pupils be contracted and the eye bright at one examination, with energy and vivacity in the whole demeanor, while dilated pupils and despondency are seen together a few hours later the patient is probably a morphin habitué. Myosis is often seen in meningitis, tabes dorsalis and other serious nervous diseases.

A well-marked arcus senilis leads to the presumption that severe



FIG. 1.—BRANCHIAL CYST.

pain about the heart is due to true angina pectoris, from the frequent association of this sign with arteriosclerosis, high arterial tension and angina. It leads one to think of cardiac dyspnea rather than pulmonary, should shortness of breath be complained of, from its frequent association with myocarditis. It should, if characteristic, cause the rejection of a candidate for life insurance, regardless of his age (for it may be seen in the twenties), since, like atheroma, it is extremely suggestive of senile changes.

In examining the ear we should note pallor or cyanosis; the "pit" left from imperfect closure of the first branchial cleft; the corns which develop upon the ears of bed-ridden patients; gouty tophi; loss of parts by freezing, bites, cuts, burns in epileptics, tearing of the lobule from earrings, etc. The thick ear of erysipelas, hematoma auris, blanching from vasomotor irritation, and redness from vasomotor paralysis in aneurism, the two conditions even upon opposite sides in the same patient, deserve attention. Tenderness, discharge, edema, deafness and tinnitus must be investigated if present. The scars from operation upon the mastoid may be noted.

Edema of the lower lids is common in dropsical diseases and after long use of arsenic, in the conjunctivitis often accompanying error of refraction and other eye conditions, and often after exposure to bright lights, especially of electric origin. Edema predominating on one side of the face is generally due either to heart disease or to nephritis, the right side being most often affected. This is due to the fact that cardiopaths generally lie chiefly upon the right side, in order to give the diseased heart a better chance to do its work. Though less constant in nephritis, it often occurs because of the concomitant cardiac changes. Edema about one eye may be due to local acute inflammation as notably in gonorrheal or other infective ophthalmias and acute inflammation of the tear duct. If exophthalmos be also present it should especially cause one to think of thrombosis of the cavernous sinus. Giant urticaria or angioneurotic edema may cause great swelling over a limited area.

When aneurism of the aorta or glandular enlargement of Hodgkin's disease, sarcoma, etc., cause pressure upon the superior vena cava, the bluish color may be intense, but with the addition of great

congestion and swelling of the tissues of the face and prominence of the valves in the veins. The neck may increase three to six inches in circumference under these conditions.

In chronic cyanotic polycythemia the blueness of the lips may resemble that of the ripe concord grape. The metallic bluish luster of argyrimism is becoming more rare, and is now practically unknown in patients less than fifty or sixty years of age, owing to the abandonment of this remedy for epilepsy and similar diseases for which it was formerly given. Cyanosis, from the use of acetanilid and other coal-tar derivatives, is very common. Steele found a cord tied about the arm to produce local cyanosis in a malingerer. The almost black cyanosis of certain cases of acute pneumonia is practically a fatal sign. Enterogenous cyanosis is decidedly rare. The xanthomatous patches, seen most often on the lower lid near the inner canthus, and frequently on the upper lid as well, and resembling strips of new buckskin attached to the normal colored skin, are seen in many cases where no especial significance attaches to their presence. In certain cases, however, they are associated with disease of the gall passages, and they should always suggest a careful examination of this apparatus.

Vitiligo of the cervical region is so commonly of syphilitic origin in young women that it should prompt a searching inquiry when found.

A red, prominent, acutely inflamed area with a black eschar should lead us to seek for the bacillus of anthrax.

The eruptions of those varieties of herpes zoster seen about the lips, eyes, and face are very characteristic, and easily recognized if such a possibility be thought of. Marked adenitis is a characteristic. Acute inflammation of the tear duct causes so marked a swelling with redness and edema about the affected region that it is often mistaken by physicians for erysipelas.

Much may be learned from observation of the hair and scalp. Premature baldness is so common as to need no description. The falling of the hair, in secondary syphilis, often more or less irregularly, verified by the abnormal ease with which hairs come away on making a slight pull, is easily recognized. The eyebrows often become



much thinned in this condition, and, at the outer angle, in myxedema. The patchy baldness of alopecia areata in young people with abundant hair elsewhere is very striking. The appearances brought about by lice with matting of the hair and enlargement of postcervical glands (*plica polonica*) are rarely seen in this country excepting in the foreign districts of a few great cities. The presence of a yellow scab and the breaking off of the hairs are characteristics of favus.

Premature grayness is often hereditary, especially if localized. Those patients who present this symptom should be carefully studied, however, since it is often the expression of nerve wear, as in young women with painful pelvic disease, patients with locomotor ataxia, etc. The rather sudden turning gray of the hair in the course of a few months in a patient of less than middle age should be regarded as a symptom of some severe stress, or of some failure in resistance, due to digestive troubles, exhaustion from pain, etc.

The dark hair near the scalp showing plainly by contrast the bleached hair, an appearance so common in sick women in hospital practice, is perhaps the most striking single sign of the moral status of the individual.

Nodules or tumors of the skin must be noted if present. Amongst others of interest we should mention the shotty papules of small-pox; the rheumatic nodules about the neck, wrists, fingers, etc., of rheumatic children; echinococcic or other parasitic cysts; multiple neurofibromata (Von Recklinghausen's disease); fatty tumors, and foreign bodies (shot).

### POSITION IN BED

The patient with acute disease, lying quietly upon the back with normal position and intelligence, may be assumed to be suffering from no very painful, and in most cases from no very serious illness. In conditions of great muscular weakness associated with dulled mental conditions, notably typhoid fever, we find the patient slipping down into the center of the bed, "lying in a heap," as it were. In advanced and serious illness the patient is often too weak and ill to make effort to turn upon the side and lies helplessly upon the back.

In acute abdominal disturbances we may see the patient lying upon the back with legs drawn up, since he finds the tension upon the abdomen and the consequent discomfort to be relieved by thus relaxing the abdominal muscles.

Because of the great frequency of appendicitis we see the right leg drawn up oftener than the left. Such patients move the head and arms much more easily than the legs, and may later present a facial expression indicative of anything between the acute and terribly painful stages of spreading peritonitis and the *facies hippocratica* of approaching death, with the cyanotic lips and pinched, anxious countenance covered with beads of sweat.

When colicky pain is present, as in gall-stone disease, stone in the kidney, acute inflammatory conditions in the intestines, in the stage of muscular cramp, and before peritonitis dominates the scene, in the various perforative abdominal lesions, extra-uterine pregnancy, etc., we see the patient writhing with pain, holding the abdomen, or lying on it by turns. If the pain is accompanied by internal hemorrhage, the patient is restless, anxious, pale, and finally cyanotic, often suffering for lack of breath. In general, colicky pain with flushed face and full pulse presents no such gravity as that accompanied by the last-mentioned signs. We may save many patients by surgical intervention if we correctly diagnose their disease before the collapse of hemorrhage and spreading peritonitis renders operation hopeless.

With acutely painful joints, notably in acute articular rheumatism, if many are involved, the patient lies almost immovable, often moving the eyes only and a heavy step upon the floor or an approach to the bedside causes an anxious facial expression, for even this slight jar of the bed intensifies the pain. We see this condition less frequently than before the introduction of the salicylates. Children with scurvy, rickets, or syphilitic pseudoparalysis often show great fear of being moved because of the pain produced.

If the pleura be acutely involved, the patient may lie upon the affected side so as to use more freely the normal lung and save himself the pain resulting from movement of the affected pleura, or there may be so much soreness that he lies upon the well side. If effusion

occurs he more constantly lies upon the side affected, since he can then use his better lung and his heart escapes the disturbance coming from the weight of the effusion.

Patients with advanced valvular disease of the heart lie most frequently upon the right side, since the dilated heart then works more easily. It is in consequence of this position that we so frequently see edema of the right hand and arm in these patients, for the veins of this extremity are compressed in the lateral position. The edema of the face upon the same side is more purely an effect of gravity, and is much more notable in those suffering from renal affections with concomitant cardiac involvement.

Many cardiac patients, and especially those with cardiac involvement secondary to chronic lung disease, are unable to lie down, and we find them in various positions from that of being slightly propped up in bed to that of actual orthopnea. By this term we mean the assumption of the upright posture because of inability to breathe otherwise. It is seen in great dilation of the heart, advanced pleural or pericardial effusion, asthma, emphysema, abdominal distention preventing descent of the diaphragm, etc. In laryngeal obstruction the patient often becomes cyanotic and clutches at the throat. The most tragic orthopnea may be seen in cases of pulmonary embolism.

In acute paroxysms of asthma we may see the patient leaning forward in such a position as to enable him to use the accessory respiratory muscles. A large aortic aneurism may compel the patient to sit up, and lean forward, or assume various constrained positions, dependent upon its size and the organs pressed upon in its growth.

The lateral position, with the knees drawn up even to the chin, may be noted in patients with cerebellar disease. In children with the chronic type of cerebrospinal meningitis, it is possible for this position to be held for months, until bed sores develop over nearly every prominent bony protuberance, and yet with final recovery. Opisthotonus occurs in those diseases presenting spasms of the muscles of the back, so that the trunk is arched with the concavity backward. In strychnin poisoning and in tetanus, the weight may rest upon the back of the head and the heels. In exaggerated forms

of hysteria seen more especially in the Latin races, a similar position is noted. The most common manifestation clinically of the tendency toward opisthotonus is that partial form seen typically in cerebrospinal meningitis and to some extent in tuberculous meningitis, and other forms in which spasm of the neck muscles causes retraction of the head.

Emprosthotonus, dependent upon spasmodic contraction of the anterior muscles sufficient to overcome their posterior antagonists, is much less commonly seen, but may be present in strychnin poisoning, tetanus, and hysteria. Pleurosthotonus, due to unilâteral muscular spasm, is less common.

Patients occasionally lie flat upon the abdomen, as mentioned above, in attacks of colic, often with a pillow under the belly. Aneurism, cancer, or other diseases eroding the bones of the spine, or visceral disease such as gastric ulcer where peritoneal irritation is present, may cause the patient to assume this rather infrequent position.

### STATION

Humpback, lateral curvature of the spine and osteoarthritis or other cause of fixation and rigidity of the spine produce characteristic attitudes. After operations upon the gall-bladder and stomach, patients frequently cannot stand erect for a time because of the pull of adhesions, and in sciatica, chronic pleurisy, abscess of lung, liver, or appendix they may bend over toward the affected side. The spine may show marked lateral curvature in duodenal ulcer.

By static ataxia we mean the loss of power to stand steadily. It is most frequently seen as a manifestation of the free use of alcohol, but may be due to sheer weakness. It is of great significance in cerebellar disease, Ménière's disease and tabes. When the patient stands with the feet touching and the eyes closed, a movement of the head in any direction of nearly an inch is allowable.

### THE GAIT

Much may be learned by observation of the gait. The peculiarities may be best noted in those cases where they are not easily ap-

parent by having the legs entirely uncovered. The patient with the heavy abdomen from any cause leans backward and spreads the feet wide apart in walking. A weak, unsteady gait, with swollen feet and legs but without evidence of pain, is common in dropsical affections. The distressed facial expression which goes with attempts to walk calls our attention to the general character of the trouble in such painful affections as rheumatism, gout, flat-foot, sciatica, metatarsalgia or other conditions in which the arch of the foot is overstrained, and in various diseases and injuries to the joints. A characteristic waddling gait is seen in the child with congenital dislocation of the hips, the body being thrown somewhat forward by the posterior position of the head of the femur.

We need only mention the toeing-in gait of the man used to walking with moccasins; the rather stiff gait of those more accustomed to the horse's back than to the ground, in whom the wearing of spurs and of high heels prevents an entirely natural swinging of the feet; the military gait; the rolling gait of the sailor, accustomed to an unstable surface; and the peculiar "setting-down" of the foot without a normal swing in those who walk upon ice, the muscles all being held in a state of tension.

The acute and temporary ataxia of the alcoholic is commonly seen. In chronic nervous diseases, however, we see the most instructive modifications of gait. In tabes dorsalis the patient feels the ground and watches it at the same time, thus supplementing the deficiency of the sensation of touch felt normally through the locomotor apparatus, which reveals the exact relations to the surface upon which he walks. The feet are raised higher than normal, thrown forward in an impulsive and somewhat uncertain manner, and brought to the ground suddenly and oftentimes heels first with a stamp. The patient sways if he cannot see the ground, and cannot walk in the dark if his disease be far advanced. As he steps from the street car we see him feel with uncertain effort for the ground, locating its exact distance with his cane before attempting to place his foot, and getting his balance before letting go of the car.

A peculiar gait is the so-called "steppage gait" of the chronic alcoholic, due to the presence of multiple neuritis, though a similar

one is seen in beri-beri and other forms of neuritis. Because of the resemblance of this gait to that of tabes, and the absence of the knee-jerk, the alcoholic multiple neuritis is spoken of as pseudotabes. The foot is raised unusually high, as if the patient intended stepping over some obstacle, this action being necessary because of the toxic paralysis of the anterior tibial nerve and the consequent toe-drop.

In the early expansive stages of general paralysis the patient may swagger into the office with a confident gait which gives one the impression of being "overacted." I have practically made the diagnosis from this feature occasionally. In later stages the shuffling, uncertain gait is far less distinctive.

The gait in paralysis agitans often suffices for the diagnosis. The patient leans forward and hurries to get his next step taken, as if in fear that he would fall forward if it were delayed, as indeed he may do. The accompanying tremor of the hands and the expressionless face are confirmatory. This hurrying gait is spoken of as festination.

Spastic gait is noted in patients with disease of the lateral pyramidal columns. The legs move as if bending at the knee were difficult, and all the muscles are in extreme tension. The toe of the shoe is often worn from inability to raise it clear, since the spastic condition of the powerful calf muscles overcomes the contractions of the extensors. Because of the virtual lengthening of the limb thus caused the foot is swung in a semi-circle. Cross-legged gait may be seen in this disease because of the spastic action of the adductors of the thigh so that the knees strike as they pass.

The "mowing gait" of the hemiplegic, the leg being swung forward like the right leg of a man using a scythe, the pelvis being meanwhile tilted, is a unilateral expression of the same nervous affection, and is generally accompanied by the characteristic swinging movement of the paralysed arm, and often by facial paralysis and aphasia, even in those thus able to walk.

A combination of the spastic and ataxic features already noted is to be found in ataxic paraplegia. In children who have suffered from cerebral lesions in infancy we have often added to the above

features club-foot and other manifestations of the lack of proper development.

In cerebellar ataxia the gait is staggering or reeling with a tendency, in many cases, according to the site of the lesion, to stagger or fall backward, forward, or to one side. When cerebellar vertigo is also present, the patient may not even be able to stand. In Friedreich's ataxia the movements are especially clumsy, the patient swaying, staggering and shuffling in his efforts to avoid a fall. In Marie's type (hereditary cerebellar ataxia), Collins states that the patient while having the reeling, uncertain gait, often walks with the body bent forward, the head backward and the feet wide apart.

Pseudo hypertrophic muscular atrophy presents a waddling gait, the patient swaying as he walks and stumbling easily, since he has not sufficient muscular strength to walk steadily or recover himself if he loses his balance. Stair climbing becomes impossible as the disease advances.

The patient with transverse myelitis, before complete paralysis, shows slow, feeble movements of the muscles of the legs, and external support is commonly needed.

Multiple sclerosis and syphilis affecting many different centers may cause a gait characterized by spasticity and feebleness, of wholly irregular type. In the gait of hysterical paralysis the leg is dragged after the body rather than circumducted as in hemiplegia. It is more frequently left-sided. In astasia abasia, seen most frequently in young women, the patient reels to and fro, since she cannot properly coördinate her muscles when standing or walking, although they may be used perfectly while lying down.

Following anterior poliomyelitis the gait is affected in accordance with the amount of damage to the anterior horns. Commonly in those remaining able to walk, one leg is affected more than the other. It is not well-controlled because of the paralysis of certain groups of muscles, and dangles more or less helplessly. The shoe on the affected side is often worn curiously because of inability to keep the foot from scraping the ground or from being placed improperly at the end of the step.

In those having spinal injuries or osteoarthritis the spine is

held more or less rigidly fixed, and the patient guards against any jar or shock by using the utmost care to avoid any inequalities as he walks.

### HEIGHT, WEIGHT, ETC.

Height and weight vary so extremely that we do not have our attention particularly called to them unless they fail to correspond with each other or with the other features of the patient's disease. Insurance companies have a general rule as to ratio, the observance of which tends to avoid the acceptance of those much under weight, who are shown by enormous accumulated statistics to be much more subject to tuberculosis and some other chronic diseases than those over weight, who are equally non-resistant in the face of severe illness. Long severe febrile diseases, such as typhoid, and diabetes and cardiac degeneration are more to be feared in the latter class. The insurance statistics show that those of medium weight and height are the best risks, but there is the utmost latitude in individual cases. The obese man of enormous appetite and unusual mental capacity, apparently exceptionally vigorous as to nutrition, and able to stand an unbelievable amount of severe strain in business, is a fairly common type. He is a better insurance risk if obesity be hereditary in his family than otherwise. Yet a wiry individual with no superfluous fat may stand the stress of life equally well, although he does not appear to do so to the casual observer.

Much more valuable to the physician is a comparison of a patient's weight with his normal. Of acute recoverable disease typhoid causes the greatest loss, and this may, as in one case I observed, amount to more than half the normal weight, though usually within the limits of 10 per cent. to 30 per cent. A steady loss occurs in many fatal diseases, such as tuberculosis, internal cancer, cirrhosis of the liver, diabetes, chronic diarrhea, stricture of the esophagus, etc., and is of the utmost significance. The loss may be offset by the accumulation of dropsical fluid, or by the development of a tumor or cyst, for which allowance has to be made. An exception to the rule of loss of weight in gastric cancer is to be noted, as in



some of my cases in that type of scirrhus cancer accompanied with marked glandular involvement and cancerous ascites. Such a patient may gain ten pounds in weight in his last weeks, although obviously failing rapidly.

The fat is chiefly subcutaneous, but in the over-fat beer drinker is found equally in the omentum, this, with the thick abdominal wall, causing the characteristic appearance of the abdomen.

The fat tissue should feel firm to the touch, for a soft, flabby condition means loss of weight, and is often associated with progressive disease. The peculiar distribution of fat may constitute a racial peculiarity, as in the case of the fat buttocks of certain Hottentot tribes, and the somewhat striking breast development, chiefly due to adipose tissue, so commonly seen in Viennese women, and referred to as "Wienerbruste."

Notable differences are found as to the development of the muscular and bony tissues, but they have comparatively little weight in diagnosis. The overdeveloped muscles of the athlete should lead us to look for cardiac and vascular trouble, for the increased strain of maintaining such hypertrophy is likely to show itself sooner or later.

## THE SKIN

### CACHEXIA

It is of utmost importance to cultivate the habit of close observation of the color and general appearance of the patient's skin, for many diseases are practically capable of diagnosis through this means alone. One should lean heavily upon the first impression gained, for it is really more valuable than the subsequent detailed study. Hence he should endeavor to have a good light at the beginning of every examination, and daylight by all means when practicable. Jaundice is not commonly recognizable in artificial light.

By cachexia we mean a modification of the normal color, brought about by anemia, malnutrition and the chronic intoxication of disease.

**Anemia.**—In ordinary types we see simple pallor. In chlorosis there is, occasionally only, a peculiar greenish shade which is pathog-

nomonic if well marked, but which cannot be described in words. There may be a false appearance of color in chlorosis rubra, the blood showing through an abnormally thin skin with a deceptive appearance of health. The peculiar yellow pallor of pernicious anemia, with the thin, lifeless lips, and seen most characteristically after middle life, almost suffices for the diagnosis.

Worry over some serious trouble, as crime or illegitimate pregnancy, may so interfere with appetite, digestion and nutrition as to produce a ghastly anemic appearance which leads to the suspicion of fatal disease. Yet I have seen it disappear in a few weeks when the cause for worry was removed.

**Malarial Cachexia.**—In those whose red blood corpuscles have been destroyed by long-continued malaria, and in whom the liver and spleen have become enlarged, a yellowish pallor, perhaps accompanied by puffiness, develops. Bronzing of the skin may occur, and the long-continued use of arsenic may add something to the changes. Actual jaundice may be present. A considerable portion of the settlers in the Rocky Mountain region during the last fifty years have been driven from the malarial region of the southwest to a healthier climate, and the cachexia we describe has been a familiar feature to western physicians.

**Cancer.**—A somewhat similar color may be seen in cancer, most notably in internal cancer, and modified by the organ affected. It is typically a sallow-appearing type of anemia, of repulsive appearance, with shades of yellow or brown which lead writers to speak of it as "dirty." When the liver is involved a greenish-bronze color may appear which is almost pathognomonic. The blackish color of melanotic sarcoma is equally so.

**Cachexia in Other Diseases.**—Syphilis, tuberculosis and most wasting diseases produce more or less well marked cachexias, but less distinctive than those mentioned. Cachexia strumipriva is seen after complete removal of the thyroid, and the pallor is modified by skin changes in the myxedematous type.

In diabetes jaundice may occur. In *diabete bronzé* the bronzing of the skin is associated with hemochromatosis, the pigment containing iron.

The modification in the color of the skin produced by racial peculiarities need not detain us. The pigmentation of the skin of certain areas, as about the genital organs and the nipple in nearly all persons, and the axillæ and abdomen in certain individuals, especially of dark complexion, and often modified in women by pregnancy, is well recognized. Tanning, freckling and burning are seen in those who have been exposed to the sun and wind. Those long exposed to tropical sun may show a very deep brownish color of the exposed parts which may persist in some degree during life.

### JAUNDICE

By this we mean the modification in the color of the skin produced by the deposit of biliary coloring matter, bilirubin and biliverdin. It varies from the lightest yellow, perceptible only in the conjunctivæ or upon pressing a glass slide upon the skin in a good light and with most careful observation, to a well-defined yellow or dark yellow, up to various shades of olive green, and even to a bronze or blackish green. The deeper forms stain the tissues of the body as well. The sallow color noted in many people with anemia or with inactive portal circulation does not affect the conjunctivæ. The sub-conjunctival fat is yellow at times without a true jaundice. Jaundice may be due to mechanical obstruction in the common bile duct preventing the bile from entering the intestine, and resulting in its absorption into the system,—obstructive jaundice. The common causes are gall-stones, parasites or other foreign bodies or inflammation, kinking, pressure from without, or scar formation from previous inflammatory processes. The obstruction may be in the smaller intra-hepatic ducts without involvement of the common duct, as in the case of abscesses and syphilitic and other growths within the liver; even though the ducts be open we may have jaundice, as in the passive congestion of the liver, in cirrhosis, in various infectious diseases, poisoning by chloroform and many other drugs, in acute yellow atrophy, etc. The jaundice in the obstructive form is due to mechanical interference with the flow of the bile after it is normally formed; in the hemohepatogenous form, formerly spoken of as hematogenous

jaundice, the bile pigments are formed within the liver cells, but are diverted from the bile capillaries into the circulation. Increased destruction of the red cells in certain infectious diseases assists the process by providing an unusual amount of hemoglobin from which the bile pigments are formed.

Obstructive jaundice may be produced by any of the following factors:

(1) Congenital deficiency of the duct,—congenital obstructive jaundice;

(2) Closure of the common, or occasionally the hepatic duct, from inflammatory swelling of its mucous membrane, generally from extension into the duct of the inflammation of a gastroduodenal catarrh,—catarrhal jaundice;

(3) Closure of the duct by foreign bodies in it,—gall-stones, lumbricoid worms or other parasites, hydatid cysts, inspissated bile;

(4) Closure of the duct by change in its form due to cicatrices, kinks, etc.,—cicatrix from ulceration produced by stone or abscess within the duct, kinks or twists of the duct due to distortion, from contraction of adhesions caused by perihepatitis, or by abscess or other inflammatory process in the neighborhood of the duct, but outside of it, or to dragging upon the duct from ptosis of the stomach or kidney;

(5) Closure of the duct from pressure from without,—pressure from enlarged glands; from various malignant growths within the liver or in the pancreas, stomach, duodenum, kidney, etc.; hydatid cysts, gummata, aneurism, lymphadenoma, tumors of the lower abdomen, etc.

We may have hemohepatogenous jaundice associated with cirrhosis of the liver, in various forms of infectious jaundice, acute yellow atrophy, syphilis, typhoid fever, pneumonia, the whole group of septic diseases, and in poisoning by chloroform, arsenic, phosphorus, snake venom, hydrogen sulphid, ptomaïns, various coal-tar products, etc., in passive congestion of the liver in the newborn, and in the so-called emotional jaundice. In this class we have jaundice from *parapedesis* of bile, which is diverted from the biliary capillaries

to the lymphatics or blood vessels, under the influence of poisons, changes in blood pressure, and other little understood factors.

#### THE DIFFERENTIAL DIAGNOSIS OF JAUNDICE

(a) Occurs within two or three days of birth, a mild, clear yellow, fading within ten days, not generally accompanied by the presence of bile pigment in the urine, nor itching.—*Icterus Neonatorum*.

(b) Occurs soon after birth, but becomes deeper; bile pigment appears in the urine; clay-colored stools; itching and scratching, from irritation of the skin by the retained biliary matters; steady failure of nutrition, or evidence that, though gaining in weight, the child is not doing well.—*Congenital Obliteration of the Bile Ducts*.

(c) Similar to (b) but accompanied by fever and septic symptoms, with progressive failure in most cases.—*Septic or Pyemic Jaundice*.

(d) History of congenital syphilis; general luetic appearance; large abdomen due to the size of the liver and spleen; liver smooth and firm, often visible in respiratory descent.—*Congenital Syphilis with Jaundice*.

(e) Jaundice in association with valvular disease of the heart, with great passive congestion of the liver (nutmeg liver), generally slight.—*Jaundice of Cardiac Disease*.

(f) Mild jaundice, chiefly before middle age, occasionally epidemic, never deeper than a fair yellow in color, often following indiscretions in diet, painless, generally accompanied by itching and scratching, acholic stools, jaundiced urine, moderate loss of weight, slow pulse, and tending to clear in from 10 to 40 days, with complete recovery.—*Catarrhal Jaundice*.

(g) Jaundice in association with cirrhotic processes in the liver, variable in Hanot's cirrhosis, less marked and more constant in alcoholic cirrhosis.—*Jaundice of Cirrhosis*.

(h) Jaundice of moderate degree coming on within a day or two after attacks of pain in the upper abdomen, and lasting days, weeks, or months, with loss of weight, acholic stools, bile-stained urine,

often repeated attacks of colicky pain, rigidity and tenderness in the upper right quadrant, gall-bladder not usually felt.—*Obstruction of the Common Duct by Stone, Round Worm, Etc.* (Either through lodgment of a stone sufficiently large to block the entire caliber of the duct, or sufficiently so if more or less inflammatory swelling of the mucous lining be added to it, or through such distention or distortion of the cystic duct or the gall-bladder as to produce secondary blocking of the common duct. This form *may* be absolutely painless from the beginning.)

(i) Coming on about as before but accompanied with repeated chills followed by high fever, attacks of pain, and deepening of the jaundice, the latter becoming lighter in the intervals.—*Ball Valve Stone in the Diverticulum of Vater.*

(j) Gradually increasing jaundice with loss of weight, acholic stools, bile-colored urine, and finally deepening of the jaundice to a dark yellow or even olive-green color; signs of malignant tumor may be present, especially in the upper abdomen, and a distended gall-bladder is often present.—*Blocking of the Common Duct by Cancer Within or Without the Duct, especially of the Head of the Pancreas; by Pressure of Echinococcus Cyst, Large Glands in the Hilus of the Liver, Etc.*

(k) The same with black tinge to the jaundice.—*Melanotic Malignant Disease.*

(l) Intense jaundice in pregnant women or those ill with serious disease, or after poisoning, accompanied by shrinking of the area of the liver (though enlarged at first) by vomiting, delirium, leucin and tyrosin in the urine, hemorrhage into the skin, and commonly by death inside of a few days or weeks.—*Acute Yellow Atrophy of the Liver.*

(m) Jaundice appearing on the second to fourth day of a slight febrile illness, often in epidemic form, with malaise, griping pains, vomiting and diarrhea, headache, pains in the back and legs, prostration, delirium, coma, enlargement of liver and spleen, hemorrhages, albuminuria, cylindruria, with occasional relapse and a moderate mortality rate, *Bacillus coli communis* or *Bacillus proteus fluorescens* frequently present in urine, though not certainly pathog-

nomonic.—*Weil's Disease (Epidemic Jaundice, or Acute Infectious Jaundice)*.

(n) History pointing toward poisoning by snake poison, phosphorus, chloroform, ether, chloral, arsenic, etc., or the toxins of certain infectious diseases, malaria, pneumonia (most often of the lower right lobe), relapsing fever, typhoid, typhus, yellow fever, etc., or of the septic diseases such as septicemia, pyemia and malignant endocarditis.—*Toxemic Jaundice*.

(o) Coming on after violent emotion, anger, fright, etc., unusual and perhaps not well established (believed to be due to spasm of muscular fibers at the entrance of the common duct into the duodenum, stasis of bile, and reversal of circulation in the cells of the liver.)—*Emotional Jaundice*.

(p) Diabetes exists and often, presumably at least, disease of the pancreas.—*Jaundice of Diabetes*.

(q) Amyloid disease of the liver is present.—*Jaundice of Amyloid Disease*.

### ERUPTIONS OF THE SKIN

The more specific lesions of skin diseases we shall leave to the dermatologist. A great number of eruptions are of interest to the practitioner through their bearing upon general diagnosis.

**Purpuric Spots.**—By this term we mean discoloration of the skin or mucous membranes due to subcutaneous or submucous hemorrhage. The black-and-blue spots may be of any size up to several centimeters in diameter.

Purpuric spots are common in certain acute infectious diseases, notably small-pox (black small-pox), measles (black measles), cerebrospinal meningitis, typhus fever and rheumatic fever, and ulcerative endocarditis; less common in typhoid, scarlet fever, vaccinia, gonorrheal rheumatism, and many of the infectious diseases, generally of a severe type.

**CACHECTIC PURPURA.**—This is often seen in Bright's disease, pernicious anemia, cancer, leukemia, perhaps especially acute lymphatic leukemia, pseudoleukemia, tuberculosis, heart disease, and

other exhaustive chronic diseases. Senile purpura, seen chiefly on the arms and hands, is a rare variety.

**TOXIC PURPURA.**—In certain rare individuals with marked idiosyncrasy certain drugs give rise to purpura. Iodin and the iodids head the list. Even a few grains of potassium iodid will cause the eruption, and it may prove fatal. Urticaria and even gangrene may be present. Mercury, quinin, belladonna, turpentin, copaiba, ergot, salicylic acid and many other drugs may produce purpura. It is often seen after snake bite.

**MECHANICAL PURPURA.**—After sudden great increase of blood pressure, commonly with obstruction to venous return, hemorrhagic spots may appear. In whooping-cough and epilepsy the manifestation is typical. The common abundance of the purpuric eruption on the legs is in a measure a mechanical phenomenon. New crops upon the legs and feet are often seen in any purpuric disease after allowing the patient to sit up or walk.

**NEUROBOTIC PURPURA.**—A few cases of purpura are known to have occurred after severe fright or other violent emotional disturbance. The cases occurring with each menstruation or in place of the menstrual flow are presumably of this nature. Weir Mitchell has reported three cases in which purpuric spots occurred near the seat of severe neuralgic pains, with muscular spasms. In sciatica, hemiplegia, tabes, multiple sclerosis and other nervous diseases purpura is occasionally seen.

**IDIOPATHIC PURPURA.**—By this we mean purpura not due to some recognized cause similar to those heretofore mentioned. J. H. Pratt gives a summary of 258 cases of purpura of which 194 were primary in nature:

TABLE OF 258 CASES OF PRIMARY AND SECONDARY PURPURA

<i>Primary Purpura</i>	<i>Cases</i>
Simple purpura .....	45
Simple purpura with arthritis.....	54
Purpura hemorrhagica .....	52
Henoch's purpura .....	43
	<hr/> 194



<i>Secondary Purpura</i>	<i>Cases</i>
Typhoid fever .....	10
Nephritis .....	8
Tuberculosis .....	7
Heart disease .....	6
Lymphatic leukemia .....	5
Ulcerative endocarditis .....	4
Rheumatism .....	4
Hepatic cirrhosis .....	3
Jaundice .....	3
Iodic purpura .....	2
Carcinoma of gall-bladder .....	1
Tumor of the liver.....	1
Alcoholic neuritis .....	1
Nervous purpura .....	1
Whitlow .....	1
Pneumonia .....	1
Arteriosclerosis .....	1
Chorea .....	1
Chronic arthritis .....	1
Pernicious anemia .....	1
Marasmus .....	1
Aneurism .....	1
	<hr/> 64
	<hr/> 258*

Small purpuric spots are designated as petechiæ, lines and streaks of hemorrhage as vibices, and extensive, more or less irregular spots as ecchymoses. The larger hemorrhages cause some elevation of the skin.

The new and especially small hemorrhages are red, but soon become purplish in color. Since the blood is extravasated under the skin, the color does not disappear on pressure. The gradual absorption of the blood pigment leads to the changing colors so familiar in the disappearance of the common black and blue spot. Gangrene and ulceration may occur, or pemphigoid blebs, over the seat of the hemorrhage.

\* Osler: "Modern Medicine," vol. iv.

It should be noted that similar hemorrhages may occur in the internal organs as well as in the skin, and they occasionally produce especial symptoms. Gangrene of the uvula, sloughing out of one eye, ulcerations of the gums, etc., have been noted.

In connection with purpura we should speak of purpuric manifestations in association with erythema, because of the bearing of the two upon internal disease, and especially rheumatism. Purpura rheumatica implies the association of rheumatic arthritis with purpura, but it is not sure that the former is anything more than the same arthritis which we find in connection with other hemorrhagic diseases. We should exclude all cases from this category in which hemorrhage has occurred from any of the mucous membranes.

In the severer types, pain, swelling and tenderness are found in several of the large joints, although the pain may be present alone. Within a day or two we find purpuric spots, and the joint symptoms may then subside. Tonsillitis is not uncommon at the beginning. The petechiæ are generally small and may be localized over the affected joints. Urticarial wheals are not infrequent, and simple erythema, *e. multiforme*, *e. nodosum*, or even angioneurotic edema may be noted.

Valvular lesions are practically unknown. Albuminuria rarely occurs, and the joints recover without suppuration or ankylosis. The prognosis is good. The rheumatic nature of the joint involvement is questioned recently, because of the rarity of previous attacks of acute rheumatism in these patients, and the absence of cardiac complications, the comparative rarity of purpura in typical acute rheumatism and the inefficiency of the salicylates.

In Henoch's purpura we also have the association of erythematous and urticarial lesions with those of hemorrhagic nature. Urticaria was present in 17 out of 29 of Osler's cases of "the erythema group with visceral lesions."

In purpura urticans we have slightly elevated purpuric lesions, or hemorrhage into the wheals of a definite urticaria, virtually a combination of purpura and urticaria in the same patient.

Osler regards purpura rheumatica and Henoch's purpura as hemorrhagic types of erythema exudativum.

**HENOCH'S PURPURA.**—In Henoch's purpura, chiefly a disease of children, attacks of vomiting, diarrhea, and abdominal pain occur, the stools and vomitus may contain blood, and arthritis and nephritis may be present. Similar symptoms are seen, however, in adults affected by the erythematous, urticarial and purpuric manifestations alluded to above and Henoch's purpura is best regarded as a type more common in children. It might better be called purpura abdominalis. Serious nephritis is more common in children, and hematuria, hemorrhages into the serous membranes, and even cerebral hemorrhage may occur. The visceral manifestations are thought to be due to hemorrhage or exudation into or under the serous coats of various internal organs, and the condition has actually been found at operation. Not only may we have colic or other abdominal symptoms, but in cases of this type, as in one of mine recently reported, headache, delirium, photophobia, retraction of the neck and fever, may lead to the suspicion that similar exudation or hemorrhage has involved the meninges. No operative intervention must be advised in any case presenting purpuric, urticarial or erythematous lesions, or having a history of their presence, without inquiring if all the symptoms may not be explained by the internal manifestations of the same processes.

Even in the face of fairly developed intestinal obstruction I have advised delay in one of these cases with eventual complete recovery. Inasmuch as intussusception has occurred with the lesions mentioned, however, we must not be over confident in such tactics of delay, but be ready to intervene when the symptoms become sufficiently definite and persistent.

**PURPURA HEMORRHAGICA.**—Purpura hemorrhagica is a more severe type, with hemorrhages from the mucous surfaces as a most characteristic feature. The cutaneous hemorrhages are often extensive, and large ecchymoses may be present. Hemorrhages may occur from the nose, gums, intestines, stomach, uterus, lungs or urinary tract. Acute hemorrhagic nephritis may occur. Erythematous and urticarial lesions are infrequent. We should probably class the afebrile cases as Werlhof's purpura, while those with high fever are of infectious origin. Many so-called cases of purpura hemorrhagica

have doubtless been cases of symptomatic purpura, complicating a definite infectious disease, notably malignant endocarditis or unrecognized cases of acute lymphatic leukemia with purpuric manifestations.

One should carefully consider these possibilities before making a diagnosis of purpura hemorrhagica. Hemorrhages may occur into the joints, testicle, spinal cord, brain or elsewhere internally. Marked anemia develops.

**PURPURA FULMINANS.**—In this rare and fatal variety of purpura occurring in children, enormous ecchymoses occur, without hemorrhages from the mucous membrane in the more typical cases. Hemorrhagic bullæ may be present. McCullum has seen a case in connection with scarlet fever, and several instances have occurred in convalescents from this disease. I have seen it in one case after chicken-pox.

**CHRONIC PURPURA.**—This disease may occur in a continuous form, or in outbreaks with intervals of health lasting over many years. Hemorrhages have been so frequent and profuse in certain cases that the disease has been confounded with hemophilia. The blood platelets may be greatly diminished. Death may occur from cerebral hemorrhage.

### REDNESS OF THE SKIN

The over-filling of the vessels of the skin produces a deeper color, this being red if the blood is perfectly aërated. This is physiological in persons of florid complexion, in blushing, and in those in whom heat, exertion, etc., have produced a hyperemia. The use of alcohol, the nitrites, belladonna and other drugs produces a similar effect. Under the continued use of alcohol the temporary flushing after drinking gradually gives way to a deeper and more permanent red, as the capillaries become dilated, in many persons, and acne often develops about the nose and mouth. A dusky redness supervenes later.

In fevers the flushed face is common, often, but not necessarily, more marked upon the affected side in acute or chronic lung dis-

eases, and in migraine. In aneurism or other cause of pressure producing complete paralysis of the sympathetic, the face may be deeply flushed upon one side, while the other may be paler than normal because of sympathetic irritation upon that side. In chlorosis rubra the thin skin and capillary walls allow the blood to show through more plainly than normal, and therefore the actual anemia is less apparent. The red face seen in carbonmonoxid poisoning (as after inhalation of water gas) is due to the cherry red color of carbon monoxid hemoglobin.

**Cyanosis.**—When the  $\text{CO}_2$  is imperfectly removed from the blood we have a persistence of its venous color, which we term cyanosis. We see it most distinctly in the lips and, especially in chronic disease, in the finger nails. When marked it gives the whole face and body a dusky tint.

Cyanosis may be due to causes, too numerous to mention here, preventing the air from entering the lungs, but equally to a multitude of causes preventing the blood from proper contact with the air or to the two causes combined. Any cause of mechanical pressure upon the air passages, stoppage of the trachea or bronchi, filling up of the alveoli with water, exudate, etc., or inability to breathe or obtain air, may produce the first variety; anything producing too slow current through the vessels of the lungs, whether this be obliteration of many of the vessels, as in emphysema, pressure upon them as in a pleural effusion, defective circulation because of heart disease, blocking of the return venous current by pressure of aneurism or other growth, or many similar causes, may produce the second variety.

In many conditions, as e. g., heart disease with advanced dropsy, both causes are active. In congenital heart disease the cyanosis may be partly due to faulty separation of the oxygenated from the venous blood, so that it is never fully of either color. Local cyanosis is produced generally by some cause hindering the return venous current, as pressure or thrombosis, in the extremities particularly. A fearful cyanosis with swelling is occasionally noted in the hands and face when the superior vena cava or certain of its branches are blocked by aneurism, especially of the arch of the aorta, or other tumor, or when an aneurism has burst through into the veins. Ex-

tensive axillary glandular involvement, more particularly in cancer of the breast, may cause marked swelling and cyanosis of the arm, often with notable venous engorgement and prominence of the valves.

Of late years we see many cases of cyanosis resulting from too free use of the coal-tar preparations, especially acetanilid, which produce methemoglobin in the blood. The continuous use of these drugs, as for chronic headache, eventually gives a purplish color to the lips almost as deep as that of the Concord grape. The drug habit may be suspected in those in whom other causes are not apparent.

Cyanosis is often extreme in poisoning by ordinary illuminating gas.

In advanced emphysema and in congenital heart disease no notable discomfort is noted in many patients, even though the cyanosis be extreme.

Because of the diminished oxygenation and the slowing of the blood current, the cyanotic surface is colder than normal. Derangements of the vasomotor mechanism, as by great cold, in paralysed limbs, in hysterical and neurasthenic individuals, and in those much exposed to the weather, may produce marked cyanosis. The blue edema described by French physicians is found particularly in hysteria.

**Hematoma Auris.**—Hematoma auris designates a condition in which free spontaneous hemorrhage occurs under the skin of the ear, seen especially in the insane. The swelling, and the reddish to black and blue color of the ear are characteristic. The ear may be shrivelled and deformed after the absorption of the blood.

**Tache Cérébrale.**—By tache cérébrale we mean the reddening of the skin of the abdomen and forehead following a stroke of the finger over the surface, or other similar mild irritation. It is of some value as a confirmatory sign in cases of meningitis or brain disease complicated with meningeal irritation.

## SWEATING

Sweating, visible moisture upon the skin, is normal under certain conditions of exertion and high temperature. In certain febrile diseases free perspiration is notable, especially in acute rheumatism,

certain cases of typhoid fever, and malarial fever. In sepsis of many forms, in typhus, pneumonia, tuberculosis, and relapsing fever free sweating occurs after a chill or after a crisis in the disease. It is of evil significance when occurring as night sweats in tuberculosis, from collapse from any cause, or after the chills of septic diseases, but is often of good prognostic import in the continued fevers, especially as the fever declines. A cold sweat is one of the most characteristic features of certain types of arthritis deformans, the skin feeling like that of a corpse after immersion in water. In gall-stone colic and other severe painful attacks the sweating may be profuse. The secretion may be bile stained, or smell urinous, and deposit crystals of urea, as in advanced nephritis and in cholera.

Localized sweating is frequently seen in the hands and feet, especially in those neurotic individuals lacking in proper vasomotor tone, but often in those otherwise perfectly healthy. The secretion is often foul smelling. The sweating about the head in rickets is so profuse as to leave a wet spot upon the pillow in many instances.

The vasomotor derangement in migraine, neuralgia, after typhoidal parotitis, exophthalmic goiter, multiple neuritis and in diseases (aneurism) causing pressure upon the sympathetic nerve, may cause a localized or unilateral sweating. Alcohol, pilocarpin, many of the coal-tar derivatives and many other drugs, produce free diaphoresis.

Anidrosis occurs in those in whom the body fluids are withdrawn through the kidneys, bowels, etc., or are not properly renewed by drinking. Diabetes of both varieties, and other diseases presenting polyuria as a symptom, profuse diarrhea, as in cholera, and continuous vomiting, as in the vomiting of pregnancy, may cause dryness of the skin. In myxedema the skin is dry, because of lack of proper thyroid secretion; and in general anasarca, because of the interference with the circulation through the skin from the tension of the dropsy. The skin dries with a greater tendency to chapping in arid climates.

Miliaria, minute blisters containing clear fluid, are found in epidemic miliary fever, and not infrequently in typhoid, acute rheumatism, acute pneumonia and other febrile diseases. These sud-

amina contain sweat imprisoned under the superficial layer of the skin. They may practically cover the entire trunk, and may be surrounded by a red zone of inflammation, then termed *miliaria rubra*.

By *chromidrosis* we mean discolored perspiration—yellow as in jaundice, or blue when stained by the *Bacillus pyocyaneus*. Hysterical patients occasionally actually sweat blood, or a blood-stained fluid—*hematidrosis*. Vicarious menstruation through the skin is called *menidrosis*.

Changes in the amount of blood and lymph in the skin produce marked variations in the appearance, especially in the face. The engorged skin in the early stages of many febrile diseases, as notably in typhus, measles, etc., in exophthalmic goiter, and in valvular disease accompanied by deficient circulation, is frequently seen. On the other hand, exhaustion, emaciation, deprivation of fluids, continuous vomiting or diarrhea, shock, etc., produce a decreased fullness of the vessels with deeper wrinkles and oftentimes pallor. The patient then looks seriously ill, in many cases, and in the *facies hippocratica* we have the final expression of these changes, as in peritonitis, cholera, shock, etc., which usually forebodes speedy death.

### DROPSY: EDEMA

When the skin and subcutaneous tissues are distended by serous fluid we have the condition known as dropsy. The fluid collects in the lymph spaces because not taken up and carried off by the lymphatics after transudation from the capillaries. The skin pits upon pressure, as the superfluous fluid is displaced by the finger tip or otherwise, and does not quickly find its way back.

Dropsy may be due to: (1) altered condition of the blood, in which it is too dilute (*hydremia*) as in severe anemia; (2) obstruction to the return of the venous current, as in mitral disease; (3) inflammation, as around the area of cellulitis; or (4) it may be of nervous origin, as in *angioneurotic edema*. Various combinations of these forms may be seen.

**Anasarca.**—General edema is called *anasarca*. The skin is full,



tense and pale, though cyanosis may be present, especially in cardiac dropsy, and the skin may be red in inflammatory dropsy. The fluid may transude through minute invisible openings in the skin. The latter then becomes sodden and oftentimes inflamed through some accidental infection. Long continued dropsy leads to a chronic thickening of the skin with less easily produced pitting.

The dropsical fluid collects not only in the skin and subcutaneous lymph spaces, but in the serous cavities. We then speak of ascites, hydrothorax, hydropericardium, hydrarthrosis, hydrocephalus, etc. Nature may provide for the escape of ascitic fluid by rupture of tissues about the navel, or a hydrocephalic dropsy may discharge through the cribriform plate of the ethmoid bone. Dropsy due to altered conditions of the blood is seen in severe types of anemia, after hemorrhage, in nephritis and chronic wasting diseases. The edema is generally diffused, and if pronounced, is accompanied by effusion into the serous cavities. In nephritis the eyelids may show the first traces but it speedily appears elsewhere in severe cases. It should be sought over the lower end of the tibiæ if suspected, since it often appears here first.

The edema of advanced acute and chronic nephritis, in which the parenchyma of the kidney is notably involved, has a fairly characteristic distribution. The face, trunk and extremities are all involved, and the serous cavities as well. The fluid tends to settle to dependent parts by gravity. We therefore see along the flanks a roll of dropsical tissue giving, in typical cases, a cross section of the body represented by the diagram, Figure 2 as compared with the normal Figure 3, but often modified by the presence of ascites. The genitals are frequently involved in the edema. The feet, legs and thighs are swollen, the skin over the sacrum and back pits deeply, the forearms are so edematous that the radial pulse is felt with difficulty. If the patient lies upon either side by preference we see that side more affected, most noticeably in the face.

In chronic interstitial nephritis, little edema is seen until the heart fails, and then generally first over the tibia.

In beri-beri, scurvy, chronic diarrhea, and notably in advanced consumption, dropsy of the feet and ankles appears. If uncompli-

cated by nephritis or local causes this edema of the feet in pulmonary tuberculosis usually forebodes death within three or four weeks.

**Cardiac Edema.**—The edema of general venous obstruction, apart from that due to local obstruction, which will be considered later, is most commonly due to heart disease interfering with the venous return, the lungs often contributing to the result. Impairment in strength of the aspiratory force of the respiratory movements, and of the length of the diastole of the heart, are prominent causes. Cyanosis is frequently present from the slowing of the circulation and imperfect aëration. The lymph flow is impeded by the same general causes that impede the venous return.

Because the dropsy is dependent largely upon derangement of mechanical forces of the circulation, it is more influenced by gravity than other dropsies. We see cardiac edema first about the ankles and lower shins, especially after a long day's work. Not until this has existed for some time does it appear in the thighs, genitals, abdominal wall, peritoneal and other serous cavities, and perhaps finally in the face and arms. The patient may work for a long time with moderate edema of the feet and ankles, but when it spreads beyond this location but little physical effort is generally possible, so that the patient speedily gives up work and goes to bed. With the change from the upright position we may find lessened edema in the legs and an increase in the thighs and lumbar

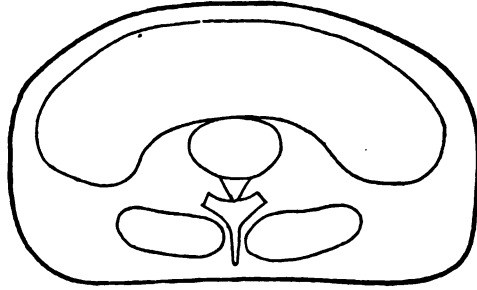


FIG. 2.—TRANSVERSE SECTION THROUGH THE TRUNK MODIFIED BY ANASARCA.

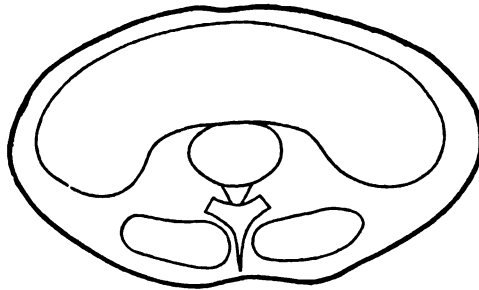


FIG. 3.—TRANSVERSE SECTION THROUGH THE NORMAL TRUNK.

region. If the patient sleeps upon one side, we find the tissues of that side engorged and perhaps even the eyelid transparent with edema. The right arm is more commonly edematous than the left in cardiac dropsy, since the cardiopath prefers to lie upon the right side because his heart is thus given more freedom for its work, and more continuous pressure is therefore exerted upon the veins bringing the return current from the upper right extremity. If one foot hangs out of bed for a time, the increased cyanosis and fullness of the skin show the tendency to stagnation in the circulation. The fluid exuding from the skin or collected in the great serous cavities is clear, almost without color, of low specific gravity, and contains no fibrin, differing in these particulars from the inflammatory exudates.

Dropsy is not wholly mechanical in origin since changes are present in the vital secretory processes, and in the endothelium of the lymph spaces.

Edematous exudation is especially abundant in certain parts, as in the genitals and the eyelids, because of the looseness of the cellular tissue there. The skin when but moderately distended is soft to the touch, but when filled to its limit becomes almost board-like. This type is seen in the legs of the patient with cardiac edema who stays upon his feet all day after a partial subsidence of the dropsy. A slight puffiness about the ankles is extremely common and without any especial pathological significance in healthy people who are on their feet all day, as are shop girls, and may occur in almost any individual after an especially long and severe day's exertion.

**Inflammatory Edema.**—By this we mean the local swelling accompanying inflammatory and suppurative processes, due to obstruction of the lymph circulation by the inflammatory exudate. The color is a faint rosy blush at first, deepening to a purplish cyanotic red as it advances. It should invariably cause the physician to look with care for some serious suppurative process in the neighborhood. Over the mastoid region its timely recognition in a semiconscious patient may lead to life-saving surgical measures. It is occasionally seen in acute inflammation of the antrum. Over the long bones it may indicate an osteomyelitis, while empyema, suppurative pericarditis,

subphrenic abscess, localized abscess within the abdomen from gall-bladder, appendical, renal or gastric disease, may first become noticeable through its presence. It is an important sign in erysipelas of severe grade, over the inflamed muscles in trichinosis, in the local manifestations of anthrax, in the swelling about the throat seen in diphtheria and scarlet fever if local suppuration is impending, in inflammatory conditions about the parotid gland, and in angina ludovici.

In thrombosis of the cerebral sinuses, because of the intimate relationship between the cerebral circulation and that of the face, edema irregularly distributed about the face is a prominent symptom. When the cavernous sinus is blocked the edema of the lids with protrusion of one or both eyes may present an almost pathognomonic picture.

**Angioneurotic Edema.**—Edema of nervous origin is spoken of as angioneurotic edema. Most typically we see it in those sudden swellings, seen most commonly about the facial region, spoken of as giant hives or giant urticaria. To some extent at least the swelling of the eyelids in arsenical poisoning and that in severe multiple neuritis are of nervous origin.

Angioneurotic edema is one of the processes leading to internal troubles in those attacks of purpura with visceral manifestations in which colic, etc., appear. If it occur about the air passages suffocation may supervene. Hysterical edema follows some ill-defined change in the nervous control of the circulation.

**Edema Neonatorum.**—A rare condition found in feeble infants exposed to cold after birth is spoken of as edema neonatorum. Hereditary edema is congenital and persistent, involves the legs chiefly, and has no well defined pathological basis.

**Local Edema.**—Local edema needs further consideration. When we find ascites before the edema manifests itself elsewhere, we think first of cirrhosis of the liver as a cause. The portal vein is obstructed, either as its branches deliver the blood from the abdominal organs into the tissues of the liver, or, because of the often associated perihepatitis, in its main current, and from backing-up of the blood current, edema occurs with transudation into the peritoneal cavity.

From pressure of the fluid upon the vena cava inferior or upon the iliac veins it is common to have edema of the legs, while the parts above the diaphragm often escape for a long time.

Edema about the face and neck is not uncommon as a result of pressure upon the vena cava superior, or the individual branches which enter into its formation, by local processes in the upper thoracic region. Thus an aneurism of the ascending aorta, or the enlarged glands of Hodgkin's disease or lymphosarcoma, may so obstruct the venous return as to cause enormous swelling, generally with cyanosis of the face and neck. The rupture of an aneurism into the vena cava may produce this condition suddenly and in extreme degree.

The blocking of the veins in an extremity from thrombosis, and especially if the lymph vessels be also occluded as is often the case, causes enormous swelling, with more or less painful edema, as in the phlegmasia alba dolens of the puerperium, and following typhoid, severe anemia, etc. The left leg is more commonly affected, but both legs may be affected after labor, and after typhoid fever and pneumonia. We may have phlebitis following small-pox, or almost any of the acute infectious diseases, and after various abdominal operations. Bacterial infection is a common but not at all invariable cause of the thrombosis.

Chronic inflammation of the veins with edema may occur as the result of infection with the bilharzia (*schistosomum hematobium*). Chronic enlargement with more or less edema of a limb or organ (scrotum) may occur from infection of the lymph system with the filaria or from a lymphangitis accompanying malignant disease, syphilis, tuberculosis, gonorrhea, etc.

### SUBCUTANEOUS EMPHYSEMA

By this term we mean the presence of air in the spaces of the subcutaneous tissue, giving rise to swelling, at first sight resembling that of edema. The lack of expected resistance to the touch, and of pitting, and the presence of a crackling feeling under the examining finger, as the bubbles of air are displaced, suffice for its distinction. In examining with the stethoscope, one must be watchful for the superficial crackling sounds to which the condition gives rise when

pressed upon, as if coarse crepitant rales were heard at the bell of the instrument.

The bubbles are usually of atmospheric air, which has gained access through some rupture of tissue communicating with the air passages, as in whooping-cough, when the root of the lung is occasionally torn by the violence of the expulsive effort; from a wound, as from fracture of a rib; tracheotomy; or from a stab injury; or from ulceration of cancer or other disease into an air containing viscus, especially the esophagus, the channel communicating with the subcutaneous surface. Emphysema is usually seen about the chest and neck, though it may spread extensively even to the fingers. It causes irregular deformity, and the parts are pale and tympanitic. The air is usually absorbed with no great trouble or delay. Air occasionally enters the tissues through the needle used for giving salt solution, etc., by hypodermoclysis. As this procedure is likely to be used in serious surgical cases, and as the physician is often called upon to examine such patients for pneumonia or other acute chest troubles; as he is called upon for similar services after fractured ribs, stab wounds, gunshot wounds, crushing injuries of the chest, etc.; and finally, as emphysema occasionally develops from infection of wounds with the *Bacillus aerogenes capsulatus* and other air-producing organisms, and the gangrenous form of infection due to the bacillus of malignant edema, it is apparent that most of the instances of emphysema will be seen in cases with some surgical features. It is of special importance that the signs of the trouble be recognized quickly, for extended opportunity for examination may not be afforded.

### LYMPHEDEMA

This term describes a swelling, usually confined to a single limb, due to transudation of lymph through the walls of the lymph vessel, usually from pressure upon or obstruction within the main vessel. When one limb becomes greatly enlarged, the condition is called macromelia. The swelling is more persistent, firm and brawny than that due to edema. The lymphedema of the scrotum and legs is often due to filarial disease (elephantiasis).

### MYXEDEMA

In this disease, originating from deficient or diseased thyroid secretion, the skin is thickened by the deposit of a mucinous substance. It is dry, rough, swollen, firm, inelastic, and does not pit upon pressure as does the skin of edema. I have known it to be mistaken for the edematous skin of Bright's disease.

### SCLERODERMA

This is a chronic hardening of the skin, as the name suggests, and it may be circumscribed or diffuse. It is especially seen about the neck and upper chest, in the circumscribed form, while the diffuse variety involves the shoulders, limbs, etc. The striking feature is that the skin cannot be pinched up in a fold as it may normally be. Motion is limited, and alterations in color are often seen.

### GLOSSY SKIN

After traumatism or certain diseases of the nerves the skin over the affected region, especially in the hands, becomes tense, smooth and shiny, as if it were varnished, and the hairs disappear. Its occasional occurrence in arthritis deformans lends support to the theory of the nervous origin of that disease.

### DESQUAMATION

The epidermis is shed after many diseases involving the skin. After scarlet fever it often occurs in large flakes, even "glove fingers" becoming detached weeks after the eruption. The cause of a nephritis may often be suspected or even definitely diagnosed by the finding of scaling on the palms and soles, especially between the fingers of the child. Small-pox, measles, erysipelas, typhoid fever, especially when treated with tub bathing, and many diseases affecting the nutrition profoundly are followed by more or less complete desquamation.

### FURUNCULOSIS

Boils are local acute suppurative inflammations, generally due to the staphylococcus aureus, and occurring around the hair folli-

cles. They indicate lowered resistance, and are often found after infectious fevers (typhoid), in those overtrained for athletic events, and in the diabetic. With the lowered resistance there is generally a special reason for the repeated infections, such as the distribution of the causative organism by poulticing and uncleanness.

### LINEAE ALBICANTES

The striations upon the skin of the abdomen due to the distention of pregnancy are well recognized, but similar striae from other causes are not considered as they should be. They occur over abdominal tumors, ascites, after rapidly developing edema, obesity or even in the convalescents from typhoid, and over the hips, thighs, breasts, deltoid muscles, etc., during the rapid growth at the time of puberty, *and in either sex*. They indicate a stretching of the skin from almost any cause, so rapid that the tissues cannot keep pace with the process.

### COLLATERAL VENOUS CIRCULATION

A study of the veins as to size, distribution, and the direction of the current may give us much information. The dilated dark veins in the aged are familiar, due to the slow current of the feeble circulation. The distention in cardiac disease, especially in those cases in which the return of the blood into the right auricle is accomplished with difficulty, may be very marked, and is associated with cyanosis and with irregular dilations of the vein due to over-distention of the valves.

If the veins be completely emptied upon deep inspiration the trouble is probably due to over-distention of the cavities of the right side of the heart. If one side remain persistently uninfluenced by respiratory movements a blocking of the innominate vein upon that side is probable. Enormous distention of the veins of the neck may occur in feeble individuals, especially children suffering from long paroxysms of coughing, particularly whooping-cough. The distention of the bulbus jugularis to the size of an egg above the right collar-bone, in whooping-cough, is alarming unless its explanation be known, but it subsides with the passing of the immediate attack.

Venous pulsation is seen as undulation, especially at the root of



the neck, not necessarily pathological, and due to the transmission into the vein of the auricular wave. In Stokes-Adam's syndrome several undulations may be noted for every arterial pulsation.

Systolic venous pulse is commonly a sign of tricuspid regurgitation, and is best seen upon the right side of the neck, though it may be present over the upper arms and chest. The veins fill from the cardiac side if emptied by pressure. Venous pulse may also be seen

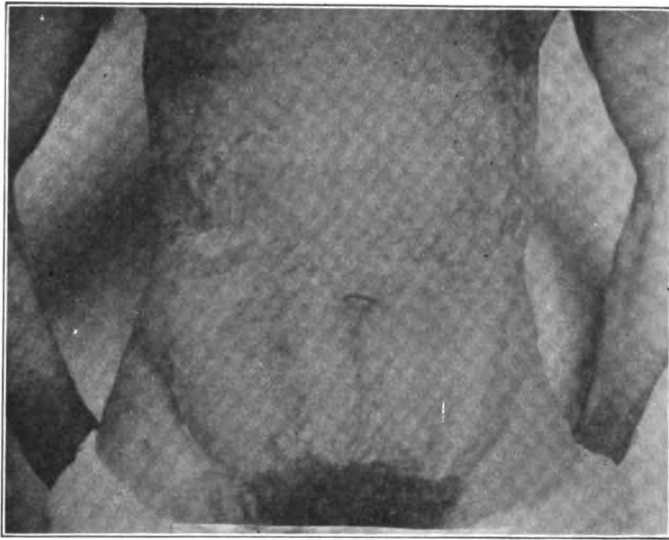


FIG. 4.—BLOCKING OF SUPERIOR VENA CAVA.

occasionally in aortic regurgitation and in the collaterally enlarged veins due to blocking of the superior or inferior vena cava.

Mediastinal tumors, often aneurismal, may compress the superior vena cava, and give rise to intense engorgement with edema and cyanosis in the face and upper extremities, as already mentioned, if the obstruction be a fairly acute one. When time is given for the development of a collateral circulation, however, we find a great increase in the size of the intercostal and internal mammary veins, and other superficial branches, for the veins must transfer the blood from the region drained by the superior vena cava to the inferior vena cava, the current of the blood flowing downward. If the inferior

cava be obstructed the process is reversed. Veins of the size of the finger may be seen over the chest and neck in such cases, and especially at the sides of the chest. Phleboliths may be found in them.

When the inferior cava becomes blocked by pressure or thrombosis, the parts drained by its branches become intensely congested,



FIG. 5.—COLLATERAL CIRCULATION IN OBSTRUCTION OF THE INFERIOR VENA CAVA.

and ascites and extensive local dropsy may be present. If the common iliac vein upon one side be obstructed, the blood from the corresponding leg is carried in the superficial epigastric and other veins to a collateral connection with the inferior cava above the point of obstruction; veins of the size of the finger carrying an upward current may then be seen. The leg remains permanently enlarged.

When the inferior cava is blocked low down, both legs may remain enormously enlarged, as in one of my cases, so as to be entirely out of proportion to the size of the individual.

When the portal vein is blocked by inflammatory conditions, or as the result of contraction of the liver or its capsule in cirrhosis of that organ, compensatory circulation is attempted by way of the superficial veins of the abdomen (*caput medusæ*) through a para-umbilical vein from the hilus of the liver, and through anastomosis of veins in the round and suspensory ligaments with the epigastric and mammary veins and their branches.

After many pregnancies the legs of women nearly always show varicosities. After a thrombosis of the veins of one leg, predominantly the left, so common after confinement, in typhoid and other fevers, and after abdominal surgical operations, the limb remains more or less permanently enlarged. Congenital or developmental weakness of the vascular walls is apparently responsible for the frequent varicosities when pressure and thrombosis are absent. Varicose veins are rare in the negro.

The enlarged venous twigs seen along the rib margin, especially in emphysematous patients, may indicate the establishment of a collateral circulation between the vessels of the lung or pleura, and those of the skin, and are especially well-marked when pleuritic adhesions are present. They are often of no significance.

### THE NAILS

One may learn something from a study of the nails as to the health and habits of the patient. Most common are the short finger nails from the habit of biting them, with the tendency of the skin to rise above the free border. The toe nails, in those careless of the toilet, become immensely thickened and distorted, for the nail growth thrown back upon itself by the resistance of the end of the shoe or the bedclothes causes this deformity.

Most significant are the transverse ridges, due to the sudden interference with the development from severe acute disease. A curved, dull-surfaced band appears at the root of the nail, and grows out at the

free edge in four or five months. If another illness, as a relapse in typhoid supervenes, another band may show above the first.

The small whitish spots which young ladies inquire so anxiously about are called leukopathia unguis, and are due to trivial injuries by which air is admitted between the cells of the nail.

In chronic disease of the chest, thickening and clubbing of the nails are common, due probably to retardation of the return circulation more than to any other factor. Absorption of fat makes the clubbing sharper and the nail narrower in tuberculosis than in heart disease. With the clubbing of the terminal phalanges we have the condition known as hippocratic fingers. They are seen in most typical form in chronic suppurative disease of the chest, especially chronic bronchitis with extensive bronchiectasis, and in empyema. Their development requires several months, and they are not necessarily permanent, in the full degree at least, for after operation in cases of empyema they may largely disappear. When the clubbing of the terminal phalanges is accompanied by thickening of the bone, we have the chronic pulmonary osteo-arthropathy of Marié. The toes are also involved, and in extreme cases the distal epiphyses of other bones of the extremities.

Cyanosis shows earlier in the nails in chronic diseases than elsewhere, and is of much significance. The combination of cyanosis and marked clubbing in tuberculosis or other chronic chest disease is an indubitable sign of the extent and great gravity of the trouble.

The capillary pulse is seen most typically in aortic regurgitation by pressing the edge of the nail lightly, so that different portions of the nail bed shall be more or less blanched by the pressure. Hemorrhages under the nail are common as the result of accidental injury, and may occur in hemorrhagic diseases. Separation of the nail often follows. The pustule of small-pox may appear here, causing decided pain.

In various nervous diseases the nutrition of the nail suffers generally in common with that of the rest of the limb. This is well seen in anterior poliomyelitis and hemiplegia. In grave organic nerve disturbances such as those of syringomyelia and various troubles accompanied by serious peripheral neuritis, dryness, brittleness,

and deformity of the nails, or even their loss, are noted. The nails are often lost in syphilis, diabetes and alopecia areata, and may be shed in hysteria from some obscure trophic disturbance. Hang nails are frequent sources of septic or syphilitic infection, particularly in medical men.

In psoriasis, eczema, ringworm, favus and rarely other chronic skin diseases the nails are occasionally affected. In acute affections, as pemphigus, exfoliative dermatitis, and erythema, the nails may be atrophied or shed. The initial lesion of syphilis occasionally affects the matrix of the nail, distinguished by the marked pain which accompanies it here.

The dark discoloration of the ends of the nails in surgeons using bichlorid of mercury, is due to the formation of the sulphid of mercury and its darkening effect upon the nail cells.

By onychia we mean inflammation and suppuration about the bed of the nail, due to varying infections, and often severe because of lowered resistance in the tissues, as in leprosy and syringomyelia. Tuberculous and syphilitic infections are common causes. The infection about an ingrowing toe nail is often severe and very painful.

## THE HAIR

The greatest variations occur in the abundance, color, distribution and texture of the hair. A lack of proper and usual hairy development is to be regarded as indicative of less than normally vigorous constitution in many cases, there being some foundation, in fact, for the common tradition that strength and well-developed hairiness go together.

With the development of the sexual functions, the hair should appear over the pubes and in the axillæ. In girls with persisting infantile uterus, it is often less developed than normal. On the other hand, in certain sexual disturbances, as in pregnancy, at the climacteric or during amenorrhea, hirsuties or increase in the hair may develop. Racial and family tendencies greatly affect the abundance of the hair.

By hypertrichosis is meant an increase in the amount of hair, and

it may be either local or general. Over concealed spina bifida a patch of long hair is occasionally seen in the lumbar region, and such an occurrence should lead to a careful examination of the spine. In children with chronic chest disease, especially empyema and tuberculosis, a growth of long silky hairs is common upon the chest. Anything causing local congestion of the skin, such as prolonged hot fomentations, as in infected wounds, or the hypodermic use of pilocarpin, with its increased sweating, may cause rapid growth of the hair.

Alopecia signifies baldness, and it may be general or local. We shall refer the reader to the works upon dermatology for its more extensive consideration. It may be of parasitic or neurotic origin. Baldness as a family disease is probably due generally to seborrhea. The partial baldness after exhausting diseases, especially typhoid, is often followed by a more abundant growth of rather darker hair. Many women lose much of their hair in child-bed. The most common cause of sudden loss of hair is secondary syphilis, all the hair of the body being involved. It commonly returns to its normal condition within a few months. The loss of the hair of the eyebrows on the temporal side is common in myxedema. Grayness of the hair, or canities, is normal at some time after middle age, but is frequently seen in the young, often in association with arteriosclerosis. Arcus senilis is often an accompaniment, and adds to the presumption of premature senility. Grayness generally develops slowly, but may come on in a few months, in those exhausted by overwork, anxiety or disease, or even in a single night from fright or terror.

Many young women with chronic pelvic disease, men with nephritis or locomotor ataxia, and patients of both sexes with serious physical trouble of any kind, become prematurely gray, although the general health may still appear to be good in some persons so affected. Patches of gray hair may be hereditary, or may develop over the distribution of a nerve affected by injury or disease, or in those suffering from migraine.

Bleached hair is easily recognized, and serves to indicate to some extent the moral status of the individual. The frequent use of

lead dyes for darkening the hair should lead us to think of the possibility of lead poisoning in certain patients. The hair is discolored in copper workers and in various industries where dyes are used.

### TEMPERATURE

Temperature is generally taken in the mouth, with the precaution that its warmth must not have been affected by the drinking of hot or cold fluids recently, and that it must be kept well closed. The rectal or vaginal temperature is more safely ascertained in infants and in delirious and other unmanageable patients, and is often  $\frac{3}{4}$  to  $1\frac{1}{2}$  degrees higher than that in the mouth or axilla. In taking the axillary or groin temperature one should be sure that the parts are wiped dry and the bulb well covered.

Fever of more than one degree may usually be detected by the touch if the hands of the examiner are not chilled or otherwise rendered less sensitive than normal. Hysterical patients and malingerers often surreptitiously and artificially increase the registration of the thermometer. Friction with the woolen blanket, with the fingers or tongue, holding against a hot-water bottle, etc., are well-known methods. When doubt or suspicion exists the physician, to make absolutely certain, should order a plain lukewarm enema to empty the rectum, and, after a few minutes to allow the temperature to become adjusted, during which time the patient should be watched to see that neither ice, hot water nor anything else is introduced, should insert the thermometer into the rectum and hold it five to ten minutes. All possibility of fraud may be thus eliminated.

Children often show a small rise of temperature, which we cannot explain. The heat centers are more easily disturbed in them than in adults.

The normal temperature is usually stated at 98.4 or 98.6 degrees F. or 37 degrees C., these figures being the average of great numbers of observations upon normal persons. Most well individuals show a subnormal temperature according to this standard for a considerable portion of the twenty-four hours. A very low morning temperature (e.g., 96° F. or 35° or 36° C.) should make us suspicious of the

existence of some disease having an afternoon rise of temperature, tuberculosis being especially to be thought of in these cases.

A very low temperature is often seen in profound collapse and after exposure to cold, especially if combined with alcoholism. The temperature may fall to 90 degrees ( $32^{\circ}$  or  $33^{\circ}$  C.) and even less with ultimate recovery, but grave danger exists in such cases. A temperature habitually subnormal, but in less degree, is seen in chronic wasting diseases, as in heart disease, nephritis, and senility. In myxedema the feeling of being cold, of which the patient often complains, is accompanied by a subnormal temperature.

Edematous or cyanotic regions show a low surface temperature, owing to the imperfect local circulation. Limbs long paralyzed or out of function from peripheral neuritis and many other nervous affections feel cold to the touch.

The surface temperature is ascertained by the use of a surface thermometer in which the bulb is a flattened coil presenting much surface to the skin. This is of most use in determining differences in temperature of the two sides of the body, as in beginning paralysis.

With the first shock of the hemiplegia a slight fall of temperature upon the surface of the paralyzed side occurs, then a rise as vasomotor relaxation takes place, and after a week or two a final and permanent fall.

Over cerebral growths and abscesses an increased temperature may be noted, and over the left hemisphere, more active in the normal individual, an increase of a degree or more is often noted, especially after mental exertion.

In paralysis agitans a local rise may be noted over the affected muscles, and in pneumonia over the affected lung. A slight rise of temperature takes place during digestion.

In most diseases the temperature may be taken twice daily. In continued fevers, especially in typhoid, when the frequency of bathing depends upon the indications of the thermometer, it is common to test the temperature every two or three hours and bathe the patient if it register above  $102.5$  degrees. As a chill is generally accompanied with sharp rise of fever, the thermometer should be used in case one occurs, and especially in septic and malarial fevers.



The slight fever in the afternoon in beginning tuberculosis and other obscure diseases must be determined by systematically taking the temperature every two or three hours if not otherwise detected.

The usual temperature chart, with record of the pulse and respiration, passage of urine and feces, occurrence of chills, sweats, etc., and with space for noting the administration of food and medicines, is used in cases of prolonged illness. The graphic portrayal of the temperature curve by means of lines connecting all the points indicated shows the physician much more of the course of the disease than a study of the individual records, and such a procedure should be carried out by the nurse when possible.

### FEVER

**Significance of Fever.**—A rise of temperature signifies that some poison circulating in the blood or some reflex irritation is disturbing the heat regulating mechanism. Fever is often a protective reaction, and should not be regarded as entirely harmful. The dilatation of blood vessels which goes with it, especially in some local focus of trouble, brings a large amount of blood to the diseased tissues, increases cellular activity and phagocytosis, and may thus aid in the development of those antitoxins upon which recovery depends. Certain bacteria are hindered in their growth by high bodily temperature. High fever may completely neutralize certain poisons, of which the best example is its well-known effect in preventing the slowing of the pulse by digitalis.

Although excessively high temperature, as in sunstroke, directly threatens life, most damage in the aggregate is done to the organism by the continued high temperature of such prolonged diseases as typhoid through the degenerative changes which result. A few weeks of fever of 103 degrees or 105 degrees do much more damage than months of the slight and intermitting fever such as we see in chronic pulmonary tuberculosis.

Fever is accompanied by increased pulse and respiration rate, and by the aching of the back and head, feeling of soreness, illness and restlessness, which we term collectively malaise. Thirst, loss of appetite, lessening of the urine, constipation and mental disturb-

ance, often amounting to delirium, are also noted. Loss of weight is one of the striking phenomena of continued fever, even though it be very mild. Chilliness or definite rigor may be noted either at the beginning of fever or during its course, especially when a marked rise occurs.

The urinary waste, especially the urea, increases during fever, and, because of the lessening of the total amount of the urine, this becomes particularly noticeable, a heavy deposit of urates being familiar in these cases, even to the laity. The suppression of the saliva and the other digestive juices has much to do with the development of the anorexia of continued fevers.

The pulse generally rises eight or ten beats per minute for each degree (C) of fever, and the respiratory rate, entirely aside from local pulmonary disease, increases about two per minute for each degree. A still sharper increase of the respiratory rate, leads us to suspect pneumonia or other pulmonic disease.

A pulse rate slower than usual in comparison with the temperature has some diagnostic significance. Thus in tuberculous meningitis the pressure at the base of the brain slows the pulse rate, while in yellow fever and certain other diseases, accompanied by jaundice, the biliary poison in the system produces a similar effect. The slow pulse seen in myocarditis may be little affected by intercurrent febrile disease.

For convenience we speak of:

	FAHRENHEIT	CENTIGRADE
Subnormal temperature, below.....	97.2	36.2
Normal temperature .....	{ 97.2	36.2
	{ 99.5	37.5
Subfebrile temperature .....	{ 99.5	37.5
	{ 100.5	38
Slight fever .....	{ 100.5	38
	{ 101.5	38.5
Moderate fever { Morning.....	101.5	38.5
{ Evening.....	103	39.5
High fever { Morning.....	103	39.5
{ Evening.....	105	40.5
Hyperpyrexia, above .....	106	41 *

\* Butler: "Diagnostics of Internal Medicine."

**Type of Fever.**—By continued fever we mean that type which remains high, with fluctuations of only one or two degrees, as in typical typhoid. Remittent fever has a larger margin of variation, but does not descend to the normal limit. It is often seen in æstivo-autumnal malaria, in hectic fever, etc. Intermittent fever descends to or below the normal line at least once in the twenty-four hours, and is best seen in typical quotidian malaria.

Recurring fever signifies a return to high temperature after several days of apyrexia, typically seen in the so-called relapsing fever, due to the spirochete of Obermayer.

Any of these types may merge into any other, or the fever may be so irregular as to be incapable of classification. The absence of the typical form of fever characteristic of a given disease is of value as a negative point in the diagnosis. Fever may be of the inverse type, the highest rise occurring in the morning, but this is of no especial significance.

Fever may begin suddenly, usually with a chill or chilly feeling, and reach its height quickly as in malaria, pyemia, etc. During the chill the skin is cool and pale from the contraction of the peripheral vessels, but it becomes flushed, hot and moist when the chill passes off and the circulatory balance is restored. When the height of the fever is attained gradually chill is generally absent, as is the rule in typhoid fever.

Fever is said to terminate by crisis when a sudden drop occurs after a certain time, generally accompanied by sweating and a return of a more normal secretion of urine. This is typical in acute pneumonia and in typhus fever. The drop may reach ten degrees in extent. The fall of the fever may be broken—an intermittent crisis.

The slow and gradual fall characteristic of typhoid fever is spoken of as decline by lysis.

In a continued fever we speak of the initial stage, during which the temperature gradually attains its full height, the fastigium or period of continued high fever, and the defervescence, during which it declines.

By relapse we mean a recurrence of the fever and characteristic symptoms, so often seen typically in typhoid, and due to reinfection.

Several relapses may occur. An intercurrent relapse begins before the complete subsidence of the original febrile attack.

By recrudescence we mean a transient return of fever not of sufficient importance to constitute a relapse, and, often at least, not due to a reinfection, but to some trifling cause, such as over-exertion or constipation. In convalescents from fevers the temperature range is more easily disturbed than in health—a labile temperature. The occurrence of fever every day, generally highest in the afternoon, is so common a clinical feature as to deserve extended consideration.

It is seen most frequently in tuberculosis and most typically in the pulmonary form of this disease, the so-called hectic fever. Chilliness may precede it. An afternoon fever of several months' duration should cause a grave suspicion of tuberculosis, and one should suspect cavity formation if under these circumstances it rise to 102 or 104 degrees with a subnormal morning temperature.

Perhaps the next most frequent cause of long-continued fever is endocarditis, and the fever may exist only when the most careful examination will show the presence of valvular lesions. In these cases, especially in malignant endocarditis, search should be made for petechiæ of embolic origin, which may clear the diagnosis at once.

Suppurative fever is found in connection with an abscess or other suppurative process. It is at times especially hard to detect the source of trouble, for an abscess in the prostate, a pus tube, suppuration in or behind the appendix, an interlobar empyema, pyelitis, obscure mastoid disease or other similar process, may not present the usual localizing signs with accustomed clearness.

Even though an abscess may have opened, imperfect drainage may still give rise to daily fever. Cerebral abscesses and cold tuberculous abscesses may present no rise of temperature.

Pyemia and septicemia generally show a high fever, that of the former being more broken in character, and accompanied by violent chills. Cabot states that the three common causes of continued fever are typhoid, sepsis of some type, and tuberculosis.

Certain affections of the liver manifest a long-continued and often severe fever. The chills, followed by high temperature so often

mistaken for ague, which accompany the intermittent infection which occurs with a ball-valve stone in the common duct, are well known. In cholecystitis, cholangitis and abscess of the liver, the fever is less sharply punctuated by chills. In Hanot's cirrhosis the fever may last for a year or two with occasional remissions of several weeks even, in its severity. The continued fever of hepatic syphilis may be accompanied with surprisingly little loss of weight.

By catheter fever we mean the sudden rise of temperature, often with chill, after the use of the sound or catheter. Most cases are probably due to some infection, though the origin is often obscure.

Malignant growths in those organs with a soft parenchyma and large blood supply, notably cancer of the liver in adults and sarcoma of the kidney in the infant, may present a temperature curve representing that of a mild continued fever. I have known two cases of cancer of the liver to be treated, by mistake, for several weeks as typhoid fever.

Slight fever is expected in the secondary stage of syphilis. In the tertiary stage it is less common, but more obscure. An unexplained continued fever should lead to the most searching inquiry for specific antecedents, a Wassermann test, or even to a trial of anti-syphilitic medication. The source may be a gumma in such a location that localization is impossible.

Certain blood diseases are accompanied by fever, the most notable one being pseudoleukemia. Acute-lymphatic leukemia runs a more febrile course than the other varieties. Anemia, and especially the pernicious form, often gives a moderate rise of temperature for months.

Ague-like paroxysms are so common in gall-stone disease, pyemia, typhoid fever, especially with phlebitis, tuberculosis, internal abscess formation, etc., that no matter what the regularity of the chills and fever the patient should not long be treated by quinin unsuccessfully on the theory of their malarial origin. Such lack of response to the treatment should lead to the most careful search for some other cause for the symptoms.

Variations in temperature may be sudden and of great degree. In sunstroke the rise from normal to 112 degrees or more may occur

in a few hours. I have seen a drop from 106 degrees to 96 in ten hours in the crisis of pneumonia, with recovery. The pre-agonal rise in acute infectious diseases may show an increase of four or six degrees in a few hours. In the chill of pyemia the variation may be still greater.

### CHILLS

The term chill or rigor applies to the sudden attack of shivering or chattering of the teeth, goose-flesh, cyanosis of lips and nails, and generally elevation of temperature, which ushers in a malarial paroxysm, an attack of some severe infectious disease, the passage of a renal or hepatic calculus, etc. The "nervous chill" of the nervously susceptible individual under excitement is not accompanied by fever nor cyanosis. In minor degree the chill is not attended with shivering nor chattering of the teeth but only by a feeling of "creepiness."

## SECTION II

### PAIN

One of the most common of the subjective symptoms for which relief is sought is pain. So important is a knowledge of its varieties and causation, and the ability to correctly estimate its severity, that the subject demands the closest study.

We judge of the severity of pain and the credit to be given it in making a diagnosis by the statements and actions of the patient and by a knowledge of the ability of the particular individual to bear pain as judged by the age, race, sex, habits, physical condition, etc. The statements and actions of the patient should fairly coincide. Nervous and imaginative persons and especially females describe as unbearable agony pain which in a stolid Russian emigrant would scarcely amount to more than moderate discomfort. The statement of the day laborer that his pain is "bad" should often receive more credence than that of a more susceptible individual that he is "suffering tortures." The great susceptibility to pain of those highly endowed, but frequently ill-balanced, persons whom we speak of as "geniuses" is well known.

Notwithstanding the statement above as to complaints of neurotic women, the female sex in general bear pain better than the male. Children endowed with an impressionable nervous system suffer more acutely in many conditions than do adults. In a rough way we may say the susceptibility to pain is great according to the greater development of the nervous system. The American Indian, the Egyptian Fellah and the Germanic and Slavonic peasants bear pain well, while the Jewish and Latin races possess a greater sensibility. Those who are exposed to open air, work and hardships in general bear pain especially well. The ability to endure it decreases according to the time of exposure, so that the bed-ridden patient complains more of a slight pain than he would have done of a much more severe one when in his accustomed health.

The condition of the mind as to its receptivity must be considered. Certain insane patients seem not to notice the presence of a carbuncle or other painful condition. Many injuries received in accidents, in battle, or under other conditions of great excitement, fail to even make their presence known until loss of blood causes faintness, or the passing of the mental excitement gives opportunity for the mind to take cognizance of the painful impressions. If shock from the injury ensue, the patient again loses his susceptibility to pain, and the absence of pain after an abdominal operation is a ground for anxiety, since it may indicate the development of surgical shock. Persons under the influence of religious emotion or various fanatical excitements often fail to give any evidence of painful diseases or injuries, resembling in this respect those suffering from acute mania.

When pain is severe the writhing of the patient, the sweat-drops upon the face, the tightened muscles and tense pulse, and the expression of agony in the face, will guide us correctly in estimating its severity. Yet we must beware of refusing to believe in the existence of pain which we cannot explain, for pain of such character does exist. Unless the patient gets relief from a placebo, or is watched when not conscious of the fact, and seen to cease his manifestations of agony, or unless he claims some distribution utterly at variance with our knowledge of the structure and function of the nervous system, we must treat his alleged pain as if it were genuine, and watch for further evidence. Nothing is more humiliating than to label a man as an imposter and later to find aneurism eroding the vertebræ, or definite signs of a cerebral tumor, which account for the complaints of the patient.

Pain is organic or functional according to the presence or absence of structural changes in the part affected. In a pure neuralgia, we regard it as functional, while in a neuritis the actual inflammation of the nerve places it in the category of organic disturbances.

Pain occurs under the greatest variety of circumstances. Many kinds are due to changes in the circulation. Thus a headache may occur from the hyperemia of over exertion, from the lack of nutrition associated with severe anemia, or from the stoppage of circulation in thrombosis of a cerebral artery. Atheromatous change lessening



the blood supply is the prime factor in the causation of the pains in intermittent claudication and angina pectoris. In inflammation the pain is in part due to the intense engorgement of the tissues.

Injuries of any kind to the tissues may cause pain, whether traumatic or due to pressure or to irritation of substances circulating in the blood, or coming in contact with the skin or mucous membranes of the body.

Pain may be reflected, as in the headache of eye-strain. In drug habitués, neurasthenics, and hysterical patients, we often cannot account for the pain complained of; although a hyper-susceptibility is present in many cases, the pain may be entirely central in origin. W. H. Thompson has described the gestures used by patients indicating different varieties of pain. They avoid touching an inflamed part, point with the finger to indicate the stabbing pain of pleurisy, or trace out the direction in which certain pains radiate, etc.

### VARIETIES OF PAIN

Dull pain is perhaps the most common. It is characteristic of inflammation of the mucous membranes and the viscera, and especially of chronic inflammation here. Parts highly supplied with sensitive nerves are more subject to other varieties of pain.

*Acute pain* is more characteristic of the inflammation of the serous membranes, of which pleurisy and the joint pains of inflammatory rheumatism are most typical. It is also found in inflammation causing acute pressure, as in felons, where pus is confined beneath the periosteum, and in the blocking of cavities, such as the antrum, or hollow viscera, as the gall-bladder, when the inflammatory products cannot escape. In neuralgia and neuritis, it may be very severe and reaches its acme in the terrible agony of angina pectoris. The erosive action of aneurisms and tumors is often accompanied by acute pain of long duration. The involvement of the sensory roots in tabes is directly responsible for the terrible lightning pains of that disease.

*Burning pain* is the especial sign of a slowly developing pressure neuritis, and is perhaps best seen in that form occurring in the inter-

costal nerves from the pressure of an aneurism of the descending part of the arch of the aorta. It is also seen in herpes zoster. The burning pain of certain neuralgias is called *causalgia*. In superficial burns, and after the application of strong irritants to the skin, burning pain is common. Throbbing pain is characteristic of acute inflammation with swelling, the pulse beat in the dilated vessels being perceptible. The action of gravity is more marked in this variety of pain than others. If the affected part is held downward the painful pulsation is greatly exaggerated.

*Gnawing pain* is perhaps best seen in cancer of the stomach or other viscera, and in tumors gradually encroaching upon other organs. Grinding and boring are epithets applied to a somewhat similar sensation.

*Aching pain* is common in the head (*cephalalgia*) and is often persistent and intense. Cerebral tumor, by increasing the intracranial pressure, causes perhaps the worst type. Intense backache occurs with the onset of acute infectious diseases, most characteristically seen in small-pox and dengue, aching of the muscles and bones accompanying it in greater or less degree. An actual chill, or a creepy sensation of cold is often associated.

*Itching pain*, as in acute conjunctivitis; shifting pain as in rheumatism; paroxysmal pain as in colic; and the varieties described as darting, lancinating, stabbing, circumscribed and localized are readily understood.

By *tenesmus* we mean the painful bearing-down pain seen in attempts (often unsuccessful) of the uterus, as in labor, the bladder, as in cystitis, and the bowel, as in dysentery, to empty themselves, and especially when they are acutely inflamed.

*Cramp* applies to the variety of pain associated with painful muscular contractions. It occurs in the skeletal muscles, as in the calves after overexertion, or in pregnancy, or when the fluids of the body have been depleted, as in cholera. Over-used muscles are subject to it, as in writer's cramp, and it is seen in poisoning by strychnin, in tetanus and tetany. A more acutely painful variety occurs in the muscular walls of the hollow viscera. It is spoken of as a colicky or griping pain, and is seen in those conditions in which the intestine,

appendix, gall-bladder, gall-ducts, kidney, ureter or stomach attempt to empty themselves of irritating contents. The pains are often intense, are generally paroxysmal, and may be accompanied or followed by fainting, vomiting, sweating, chill, collapse or even death. The pain is often relieved by the passage of gas, feces, etc., and sometimes by pressure.

*Simulated Pain.*—Malingerers oftentimes complain of pain to avoid army service or other disagreeable duty, and hysterical and neurasthenic individuals, almost unconsciously at times, probably exaggerate slight discomfort into terrible pain. Our only safeguards consist in careful and prolonged observation, in a study of the nature and severity of the pain as compared with known genuine cases, and careful scrutiny of its anatomical distribution.

*Pain in connection with acute inflammation* is commonly made worse by pressure, or in other words, tenderness exists. Movement of the affected parts, mechanical irritation, damp weather, a fall in the barometer, application of cold, expectant attention, suggestion and various other agencies may make pain worse, while rest, heat, mild climate, diversion of the attention into another channel and various emotions may render pain less severe.

### DIAGNOSTIC SIGNIFICANCE OF PAIN

Pain is ordinarily assumed to indicate disease of the part affected, but this is not necessarily the case. It may be felt at the end of a nerve when the source of irritation is more centrally located, as in the knee pains of hip disease. It may be reflected out upon another branch, as when toothache in the lower jaw causes pain in the distribution of the branch of the trifacial supplying the upper jaw. A tumor pressing upon a large nerve near its origin may cause pain over its entire area of distribution. Painful diseases of organs above the diaphragm may cause severe reflected pain below in the abdomen, so that the appendix has even been removed when the only disease present was pneumonia. The pain over the lower left ribs in the back in ulcer of the stomach, over the mid-dorsal vertebræ in gastritis, under the right scapula in gall-bladder disease, and the pain in the

left arm and even in the left side of the neck and the ear in angina pectoris, are well recognized.

By the term *allochiria* we designate the feeling of pain in an anatomically symmetrical part, as seen occasionally in *tabes* and other nervous diseases.

We often find that disease in one of a number of organs closely associated in function causes pain in other organs. The pain in the course of the ureter, and in the penis and testicle in renal colic, is an example, the nerves supplying these parts being closely associated in origin. In the pain in the breasts in association with menstrual pain we have an example in which the nervous supply is diverse.

In meningitis and other diseases of the brain and spinal cord, pain may be felt in the back, limbs, joints and muscles.

Disease of internal organs is frequently the cause of pain referred to certain neighboring superficial areas possessing a much greater degree of sensibility, and innervated from the same spinal segment. The reference of pain in hepatic colic to the epigastrium may be taken as an example.

### TABETIC CRISES

Dependent upon the pathological changes at the origin of the affected nerves we have attacks of pain referred to distant organs supplied by them. They are sudden, paroxysmal, intensely severe, and yet unaccompanied by any especial pathological changes in the affected organs. Laryngeal, pharyngeal, gastric, cardiac, rectal, urinary, intestinal and genital crises are described. Of these, gastric crises are the most common and the most important, since they simulate gall-stone or other local disease in the abdomen, and operations have been mistakenly done for their relief.

## PAIN IN DIFFERENT REGIONS

### THE HEAD

**Headache.**—Headache is so common and oftentimes so important and so readily misinterpreted that it demands especial consideration.

We distinguish it chiefly by its diffuseness from neuralgia of individual nerves of this region. Sick-headache or migraine is a neurosis with periodical manifestations which we shall consider elsewhere.

**CAUSES OF HEADACHE.**—The most common causes of headache are those associated with changes in the blood-supply of the brain and meninges. Under-filled vessels, as after hemorrhage and in anemia, fail to supply the nutritive needs of the brain, and headache often results. Arteriosclerosis with narrowing of the calibre of the cerebral vessels, and thrombosis with complete closure of them, are likewise causative factors. Increased tension, temporarily after violent exertion or after severe cough, and more or less permanently in nephritis and in many cases of arteriosclerosis, is a common cause. The circulation in the blood of various poisons, as after breathing impure air, in uremia, alcoholism, gout, diabetes, in gas poisoning, etc., should be mentioned. The sudden dilation of the vessels, after inhaling the nitrites, whether given medicinally, as nitrite of amyl, or inhaled in various occupations in which nitro-glycerin compounds are used, as in mining, shooting, manufacturing nitro-powders, etc., causes severe throbbing headache.

The specific infectious diseases are accompanied at the onset by headache, often for days together, as notably in typhoid fever. In cerebrospinal meningitis it may be of extreme severity. Inflammatory processes within the cranium, whether involving the bone, periosteum, meninges or the substance of the brain, often cause cephalalgia. In tuberculous meningitis the headache may come in severe paroxysms and last for weeks together in certain subacute cases. In this form vertigo is often present and nausea and vomiting accompany it. All these symptoms are frequently associated with the headaches of tumor of the brain, abscess of the brain, inflammation extending from the mastoid, arteriosclerotic headaches, etc. The occurrence of drowsiness and irritability render the outlook more serious in headaches accompanied by vertigo, nausea and vomiting; and the development of optic neuritis, paralysis of eye muscles, inequality of the pupils, hemiplegia, staggering gait or definite localizing symptoms leads to the decision that the cause of the headache is organic and not functional. The pressure from the growth of a tumor or abscess is the

cause of prolonged and intense headache in many instances. In hysteria, neurasthenia, epilepsy, exophthalmic goiter and in certain cases of parietic dementia and melancholia, it is a troublesome feature. Excessive fatigue, especially mental, is often accompanied by headache.

In many cases we have this symptom as a reflex phenomenon from disease of the eye, as in myopia and astigmatism; of the ear, as in otitis; of the nose, as in acute rhinitis and adenoid disease and disease of the accessory sinuses; in indigestion, notably in hyperacidity; and in disease of the genito-urinary tract, more especially in hysterical women and neurasthenic men.

Headache from eye-strain affects the frontal region most commonly, but may involve the temporal or occipital regions. It is aggravated by overuse of the eyes, as in sewing, reading fine print, etc. Patients are not necessarily aware of any difficulty in vision. The headache of glaucoma is often of great violence.

The reflex pain from nasal disease may be felt over the forehead, in the temporal region, or at the vertex. The headache from digestive troubles may be directly associated with sharp hyperacidity, or may be toxemic, as in the ordinary bilious attack.

Various drugs cause headache, among which are opium, chloral, the salicylates, alcohol, coffee, tea, tobacco, etc. Following the cessation of the use of opium, chloral and other drugs to which the patient is addicted, we may have chronic headache, relieved only by a dose of the disused drug.

The diminished atmospheric pressure of high altitudes, especially above 10,000 feet, and particularly under conditions of severe bodily exertion, often gives rise to intense headache, one of the symptoms of mountain sickness.

Syphilis may be accompanied by headache, as a secondary manifestation, or arise from changes in the vessels, meninges, periosteum, or bone, or from the development of a gumma, as a tertiary phenomenon. This form of cephalgia is generally worse at night. Neurasthenic headaches are worse upon awakening, and wear off during the day. Malarial headache, and especially the localized "brow-ague" may be definitely paroxysmal. After sunstroke and brain injuries, headache

is oftentimes prolonged, severe, paroxysmal, and, at times, accompanied by various forms of mental derangement.

In jaundice and paroxysmal hemoglobinuria headache is frequently noted. Poisons are often formed, as in acute indigestion, which, not being destroyed by the liver, circulate in the blood stream, and headache, vomiting and drowsiness ensue. When indigestion and constipation are present, relief is only obtained upon free purgation, and especially after the use of calomel.

Migraine, or sick headache, is a definite paroxysmal neurosis, in which, after various preliminary symptoms, unilateral headache develops, with prostration, nausea, vomiting, and often disturbance of vision, aphasia, or hypersensitiveness of vision, hearing, taste, smell and touch. The characteristic features of the headache are its paroxysmal nature, its unilateral character, and its severity.

In the scalp we may have myalgic or rheumatic pains with tenderness upon pressure, increasing upon voluntary movement.

**Neuralgia.**—Neuralgic pains may affect certain regions of the head, and should be differentiated from headache. Neuralgia affects rather the trunk or main branches than the peripheral distribution of the nerve, and is generally more definitely localized than headache.

The most frequent form of neuralgia is tic douloureux, or neuralgia of the fifth nerve. The pain is severe, confined to one side, generally spontaneous in origin and occurs in paroxysms, often of the utmost violence. With these violent attacks there is often reflex spasm of the facial muscles, tic convulsif. The pain is generally distributed over the side of the face. Degenerative changes in the Gasserian ganglion are present.

Neuralgia of the ophthalmic branch affects the brow and the region above the eye. It constitutes the so-called "brow ague" and is not infrequently of malarial origin. It is often accompanied by tender points as at the supra-orbital foramen and the side of the nose. Increased discharge from the nose and congestion of the conjunctiva may accompany neuralgia of the superior maxillary division of the fifth nerve. One should be careful to exclude antral or other sinus disease in these cases, for it gives rise to both these symptoms. If the outlet of the antrum be obstructed the pain is intense. As soon

as thickening occurs in the lining mucous membrane, the darkening is seen upon transillumination with the electric bulb held in the mouth. Tenderness often exists over the antrum, and the discharge, more abundant when the head is turned to the opposite side, often irritates the nostril of the affected side. Inflammation of the frontal sinuses causes pain in the forehead, and the overlying tissues are tender to percussion.

If the inferior maxillary division is affected, we have pain in the cheek, lower lip and tongue. If one branch be involved the others are likely to become so later. Arteriosclerosis is commonly present in the vessels of the region. The nerve trunks are tender. Taste is not involved. Herpetic eruptions or other trophic changes may occur.

**Pain in the Ear.**—Pain in the ear is most common as a result of acute disease of the middle ear, and is very frequent in children. It is aggravated by pressure at the tragus, and by pulling the external ear. The mastoid should be frequently percussed and examined for edema, since it is often involved in these cases. The pain is often relieved instantly by natural or surgical perforation of the drum membrane, with consequent relief of the tension.

**Pain in the Eye.**—Pain about the eye, apart from the neuralgia of the fifth nerve mentioned, is commonly due to local disease,—conjunctivitis, glaucoma, etc. Pain about the mouth is found in various local affections, stomatitis, tuberculosis, or syphilitic disease, cancer, and various suppurative conditions, disease of the teeth, etc. The cause is usually apparent upon examination.

## THE BACK

**Rachialgia.**—One of the most common of all symptoms is the backache attending the onset of acute infectious diseases. It is most severe in variola and epidemic meningitis, but in dengue, influenza, and ordinary tonsillitis may be very distressing. Headache is also present, and soreness and aching in the limbs and trunk.

Pain in the upper spine is common in hysterical and neurasthenic women. Between the scapulæ we see it in gastric disorders. The lower dorsal and lumbar regions ache from fatigue. Sacral back-



ache commonly indicates pelvic disease, of the womb, tubes and ovaries, in women; and of the bladder, prostate and especially seminal vesicles, in men. Hemorrhoids may cause it in either sex. The significance of this type of pain, particularly in men, is not properly appreciated.

Acute pain occurs anywhere in the spinal region from meningitis, being local or general in accordance with the distribution of the causative inflammation. Muscular spasm accompanies it. The pressure of meningeal hemorrhage, local irritation from development of a tumor, or from limited traumatism, may cause a very sharply localized pain in the back.

**Lumbago.**—Muscular pain in the region of the loins is very common. It is increased by movement of the affected muscles, and is bilateral. Lumbago should be differentiated from the acute pain following sudden overstrain, which is often due to rupture of muscle fibres or their attachments, and is often unilateral.

**Osteoarthritis.**—An extremely important point lies in the differentiation of lumbago, and other troubles of only moderate importance, from the more severe and disabling osteoarthritis, so commonly overlooked unless especially sought for. Pain upon movement of the spine is much more constant and severe, and limitation of motion is readily found if the patient be requested to pick up an article from the floor, the bending required taking place more at the knees and hips, and less in the spine than is normal. Forward, backward and lateral movements are painful and limited. Spasm of muscles is often to be detected. Jarring of the spinal column by rising upon the toes and dropping suddenly upon the heels, is rather less painful than in caries from tuberculosis, syphilis, traumatism, or pressure from aneurism, cancer, etc., and deformities are less sharply localized. Pain extending along the intercostal and lumbar nerves is frequent, and generally more diffuse than in caries. Tenderness over the sacro-iliac joints is found in local disease of that region, and is aggravated by jarring or motion. Relaxation of the ligaments, syphilitic and tuberculous disease, and acute infectious arthritis, as in sepsis, gonorrhea, and so forth, are to be considered.

The relief obtained from the application of a supporting jacket

is confirmatory evidence of the diagnosis in osteoarthritis and caries of the spine.

**Typhoid Spine.**—The so-called typhoid spine, although often having a neurasthenic basis and formerly believed to be of neurotic origin, is accompanied at times by definite bony and periosteal changes demonstrable by the X-ray, as shown in some of my cases, by Drs. Packard and Stover. This means of examination should never be neglected in persisting painful disease of the spinal column.

**Diseases of the Spinal Cord.**—Diseases of the spinal cord are more likely to give rise to pains in the line of distribution of the nerves, as in the lightning pains of tabes, or the girdle pains of myelitis, and are not accompanied, unless by accident, with bony disease of the spine. The changes in the knee-jerk, presence of paralysis, contractures, etc., are further signs to be sought in disease of the cord.

### THE CHEST

Pain in the chest may be due to purely superficial irritation, as from the application of croton oil, or sinapisms. Herpes zoster gives rise to a unilateral pain, and even before the eruption appears the parts affected are tender. The patient flinches from the touch of his clothing in severe cases.

Structural damage to the bony, periosteal, cartilaginous or muscular tissues of the chest is generally readily detected. It is of the utmost importance to inspect the chest in a good light, since the localized bulging of periostitis, gumma, aneurism, empyema, hernia of the lung, or the projecting tumor of pseudoleukemia or malignant disease may otherwise escape detection.

Pain in the chest is frequently caused also by affections of the contained viscera. Inflammation of the serous membranes, pericardial and pleural, gives rise to sharp sticking pain, the latter more influenced by respiration, and both exaggerated by pressure. Serous and plastic inflammations here are more painful than purulent ones, the purulent secretion acting as a lubricant between the inflamed serous surfaces. Pleural pain is felt anteriorly more than posteriorly. Diaphragmatic pleurisy often gives rise to excessively sharp pain, greatly exaggerated by upward pressure beneath the border of the

ribs, and often reflected into the abdomen. It thus simulates appendicitis and other acute abdominal infections, and should be carefully guarded against in differential diagnosis. Pneumonia, abscess and gangrene of lung give little pain unless the pleura be involved, when the pain is as above described under pleurisy. Pain from dry pleurisy is very common in pulmonary tuberculosis and is often upon the comparatively healthy side, because of previous obliteration of the other pleural cavity. Pleuritic pain and friction low in the left side, after abdominal inflammations, often signify a pleurisy by extension from a subphrenic abscess, such as results from perforation of the gall-bladder, of gastric ulcer or pancreatic abscess into the lesser peritoneal cavity. I have seen it in all these conditions, and consider it a sign of great value.

Large pericardial exudates may give rise to sharp pain, although often rather to a feeling of distressing distension, rendered worse by pressure upon the tip of the sternum.

Acute bronchitis gives a very characteristic feeling of pain and soreness under the sternum, much exaggerated by coughing.

Excepting in aortic regurgitation pain is not common in the valvular affections of the heart, nor is it a feature of acute endocarditis. In aneurism of the thoracic aorta, pain is a prominent symptom. It is most constant and severe when erosion of the vertebræ is taking place in aneurism of the posterior and descending portions of the arch.

When the disease affects the ascending portion the ribs are frequently eroded, but the distress is less than in erosion of the spine. When the intercostal nerves are pressed upon, notably in aneurism of the descending thoracic aorta, the pressure neuritis gives rise to a burning pain along certain intercostal spaces of the left side which is almost pathognomonic. The sudden partial giving way of parts of the wall of the aneurism is accompanied by severe lancinating pain.

In mediastinal tumor pain is much less constant and severe, and in mediastinal abscess, more throbbing in character, than in aneurism. Acute aortitis may present pain under the sternum.

The pain of angina pectoris is perhaps the most severe of any known. It is felt through the breast (breast-pang) and radiates to

the left shoulder and arm and occasionally to the left side of the neck and ear or to the palate. It is of such character and intensity that the subject stands still in agony, unable to breathe except in the most superficial manner. Pallor accompanies it, and sudden death not infrequently occurs during the paroxysm. One of my cases died thus in my presence during the first moments of his first attack.

Pseudo- or hysterical angina has only superficial resemblances. The pain is doubtless severe, but has not the sensation of impending death of true angina. The patient tosses about, cries out, and acts as any hysterical patient might do. The one condition is accompanied by the whole group of phenomena: age, arteriosclerosis and increased blood pressure, which signifies a wearing-out of the circulatory mechanism; the other by the ordinary conditions of hysteria, neurasthenia, or other functional neurosis.

#### THE SIDE

We have noted the pain of pleurisy and the other conditions of which pain in the side is a part. Local inflammation, tumors, peritonitis, and injuries as causes of pain are readily detected.

Pleurodynia, or muscular rheumatism, is less frequent than lumbago and has the same characteristics. Affecting the intercostal muscles it makes a full respiration impossible because of the pain upon motion. Tenderness is less notable than in intercostal neuralgia, and is diffuse, while in the latter we have the well-defined points of Valleix, particularly under the tip of the scapula, in the midaxilla, and under the breast upon the left side. The tenderness above the cardiac region so often complained of, and believed by the patient to indicate cardiac disease, is due to this cause. It is so common in those using tea, coffee, or tobacco to excess, that it deserves careful consideration. Why it is predominantly left-sided remains unexplained.

Muscular affections as tetanus or trichinosis, may cause pain in the side.

Side pains may be due to disease of the organs within the abdomen, as in abscess of the liver, sub-phrenic abscess, an enlarged spleen (from malaria, leukemia, etc.) appendicitis, especially when

the appendix is located well to the outside of the cecum, nephritic abscess, pyelitis, perinephritis, nephritic colic, sarcoma of the kidney, especially in babies, hepatic colic, over distended colon or cecum, even from chronic constipation. Attacks of strangulation of a floating kidney, called Dietl's crises, give rise to severe pain, paroxysmal and radiating along the course of the ureter to the bladder or to the genital organs.

Sudden distention of the capsule of the liver or spleen gives rise to acute or aching pain in the side affected. The acute pain from swelling of the spleen in boys after running is thus accounted for. The liver may swell so much even in catarrhal jaundice as to cause such pain, sometimes on the left side, from swelling of the left lobe. The sudden appearance of pain over the liver, spleen or kidney region in malignant endocarditis should lead us to suspect that an infarct from embolism has taken place. Peritoneal friction may be found over the spleen or liver as a confirmatory sign. Such friction is frequently found over the spleen in leukemia, and over the liver also in lymphatic leukemia. The development of secondary malignant nodules in these organs may give rise to similar pain.

Neuralgia, or pressure neuritis may cause pain in the side of the abdomen as in the side of the chest.

### THE ABDOMEN

Pain in the abdomen is probably more important, all things being considered, than that in any other part of the body, since many of the affections of the abdominal organs accompanied by pain are acute and dangerous ones demanding more or less immediate surgical relief, and pain is often our best guide to the diagnosis. We shall consider at some length the varieties of pain associated with diseases of the different organs in the appropriate sections. Probably most acute pains in the abdomen come from perforation of some of the hollow viscera, or some obstruction of their calibre by a foreign body. When ulcer of the stomach or duodenum, typhoid or other ulceration of the bowel, acute appendicitis, gangrenous gall-bladder, pus tube, extra-uterine pregnancy, cancer of any hollow organ, aneurism, etc., become perforated, and the contents of the viscus enter the peritoneal

cavity, the pain is often as sudden and as severe as that of a gunshot wound. The appearance of shock, the rapidly developing rigidity of the abdomen, the rise in blood tension and concomitant wiry pulse, and later the appearance of fluid in the abdominal cavity, of friction from the inflamed and roughened peritoneum, the development of the hippocratic face, and the rapid approach of death (generally within twenty-four to forty-eight hours) point certainly to the diagnosis.

The blocking of the gall-duct or neck of the gall-bladder by gall-stone, of the pancreatic duct by pancreatic stone, of the ureter by kidney-stone, of the bowel by foreign body, kink or twist, intussusception or other cause, or of the appendix by kinking, swelling, etc., gives rise to acute colicky pain, but without the shock and other features of perforative accidents. The pain of a blocked fallopian tube and that of the uterus attempting to expel its contents are somewhat similar.

The pain of cancer of the abdominal organs is likely to be dull and constant, excepting that when the peritoneum is involved, as over a cancerous nodule in the liver, the pain is very acute, and much aggravated by pressure. The pain of gastric or duodenal ulcer varies from the usual dull, burning discomfort to the acute pain of peritoneal irritation if perforation be imminent. Although temporarily aggravated by food, it is often worse when the stomach is empty.

Subphrenic or hepatic abscess or other suppurative process gives rise to a dull, aching pain, occasionally throbbing in character, or sharp from peritoneal involvement. The pain of acute pancreatic disease is intense, and often accompanied by extreme shock. Aneurism gives a dull steady pain, similar to that of cancer but sometimes throbbing, often a boring pain if bone be eroded, or burning if nerves be pressed upon. The angina abdominis associated with sclerotic vessels is an intense pain located in the epigastric region, and is often wrongly diagnosed as gastralgia.

A small omental hernia through the linea alba, or adhesions between the stomach and the anterior abdominal wall, may give a constant nagging, dragging pain, which eventually makes an invalid of

a strong man. Other adhesions cause pain chiefly as they interfere with the functions of the organs involved. Myalgia of the abdominal walls and neuralgia of the lumbar nerves have the same characteristics that similar affections have elsewhere. Dull pain over the liver, with colicky exacerbations, is seen in Hanot's cirrhosis. The gastric crises of locomotor ataxia are generally intensely painful, and often accompanied by vomiting of very acid gastric secretions, but pain may be wanting.

Gastralgia was formerly diagnosed frequently. I have seen a great number of abdomens opened because of pain, but have never found it necessary to explain the cause as being gastralgic, because some definite pathological lesion has always been present. In arsenical neuritis a definite gastralgia may be seen, but the diagnosis in any case should be scrutinized with the most searching care, and even then is probably rarely justified.

Dull pain under the sternum is seen in spasm or cancer of the esophagus and cardiac end of the stomach, and in the epigastric region in chronic gastritis.

Lead colic, the "dry bellyache" of the laity, is paroxysmal and severe, and felt chiefly at the navel. Girdle sensations are seen in certain nervous diseases, especially in myelitis.

The referred pain in the right side of the abdomen in acute disease of the lung, formerly so often mistaken for appendicitis, comes from the reflection into the abdomen of the pain of the diaphragmatic pleurisy so often associated with acute pneumonia. I have seen it referred to the appendical region from such a lesion in the lower *left* lobe. It is possible for the original source of such referred pain to be far away. Thus following an esophagotomy, a mediastinitis developed, and I was told by the attending physician that very acute pain in the lower left abdomen had supervened. Stating that it was probably to be explained as a referred pain from a left-sided pleurisy by extension from the mediastinitis, we examined the chest, and found dry friction over the whole lower part of the left lung, abundantly confirming the diagnosis.

The ordinary griping pain from the sharp peristalsis caused by drugs or by flatus is familiar to everyone.

When the rectum and sigmoid flexures are inflamed, a bearing-down pain is complained of, with tenesmus.

### THE EXTREMITIES

**Upper Extremities.**—We have referred to the pain shooting down the left arm in angina pectoris, the ulnar distribution being chiefly involved. Occupation neuroses, of which writer's cramp may be taken as a type, give a cramping pain in the muscles affected, especially upon attempted motion. Local inflammatory conditions give pain in the hands oftener than in the arms. (*See below for consideration of pains felt in both upper and lower extremities.*)

**Lower Extremities.**—In the lower extremities we have the pains of sciatica, neuralgia or neuritis of the sciatic nerve; the lightning pains of tabes; pain in the knees referred from hip-joint disease and obturator hernia; pain in the leg and foot from flat-foot, so often treated as rheumatism; that from varicose veins and ulcers; metatarsalgia, from pressure upon one of the plantar nerves by the head of the corresponding metatarsal bone; painful cramps of calf muscles, as in pregnancy and in swimmers; painful gonorrheal exostoses on the plantar surface of the os calcis; pain in the large joint of the great toe and elsewhere, from gout; pain from traumatic neuritis; from bunions; from corns; from ingrowing toenails; etc. In the knee we have acute pain, often causing an instantaneous fall, from a loose cartilage. Painful cramp is also seen in arteriosclerotic subjects from intermittent claudication, coming on after effort which is too great for the ill-nourished muscles of the individual, and passing away upon rest.

**Upper and Lower Extremities.**—We have pain from many affections common to both upper and lower extremities, and involving any of the tissues. Traumatism needs only to be mentioned. Unusual exertion leaves the muscles stiff and sore. The muscles of the extremities in common with those of the trunk suffer in myositis and especially when infested with the larvæ of the trichina. Children with rickets and infantile scurvy dislike to be moved because of the pain and tenderness in the muscles and joints.



Many painful conditions are directly associated with disease of the blood vessels. The sudden pain from the lodgment of an embolus at a point of narrowing in the artery, followed by loss of pulsation below, and subsequent signs of the absence of circulation, is characteristic. Arterial thrombosis, as occasionally seen in typhoid and in arteriosclerosis is less painful. Venous thrombosis is often accompanied by pain which appears before the obstruction is discernible, generally in the calf of the leg. When the obstruction is extensive and probably especially when the lymphatics are involved, the affected limb is very painful, as in phlegmasia alba dolens.

The most common painful affections of the limbs involve the nerves. We may have simple neuralgia, brachial or sciatic, or affecting certain branches only, with definite tender points, pressure upon which aggravates the pain; or a neuritis, with swollen and tender nerve trunks, or branches—toxic, rheumatic, traumatic, etc., or neuromata, sometimes multiple, or the condition described by von Recklinghausen, multiple fibro-neuromata. If bilateral, pains of nervous origin are commonly due to spinal disease or some affection of the cord, excepting in multiple neuritis.

Aching neuralgic pain is occasionally seen in the affected leg in adults with a beginning anterior poliomyelitis. I have known such a pain to be treated for weeks for sciatica. The toxic neuritis due to lead affects particularly the distribution of the radial branch of the musculospiral nerve; that from arsenic affects the forearms and the legs, is more painful, and the muscles of the arm and leg are very painful upon pressure; the vagus nerve may be involved with fatal result. The neuritis from alcohol, by far the most common, affects both upper and lower extremities, with a preference for the extensors. The distribution is the same after acute infectious diseases, typhoid, typhus, influenza, gout, rheumatism, diabetes, small-pox, syphilis, tuberculosis, malaria, etc., and the pain is in general less prominent than in the forms following mineral and other poisons coming from without the body. The multiple neuritis after carbon monoxid poisoning, as in inhaling illuminating gas, is more sensory than motor, and accompanied by paresthesia rather than by severe pain. In that of leprosy anesthesia is more prominent than

pain, and in beri-beri pain is not a prominent symptom. The limbs are far less often affected in diphtheritic neuritis than is the distribution of the vagus. The mild form of neuritis, known as tender-toes, in typhoid fever, seems to follow cold tub bathing very frequently, and is characterized more by tenderness than pain. It is said to be accompanied by local venous thrombosis.

**JOINTS.**—Pain in the joints suggests at once the possibility of acute articular rheumatism, with, usually, symmetrical involvement, redness, swelling and exquisite tenderness. Effusion is frequent in the larger joints. Gout affects most frequently the metatarsophalangeal joint of the great toe, and the red, shining skin covering the swollen tissues is fairly characteristic. The other joints of the foot and those of the wrist and hand may be affected.

Arthritis deformans may be so acute as to be mistaken for acute rheumatism. The pain comes usually in attacks, perhaps months apart, and the deformity gradually increases. Many cases have no acute pain.

Gonorrheal arthritis and the septic joints seen in scarlet fever, cerebrospinal meningitis, pneumonia, typhoid, dysentery, septice-mia, pyemia and septic endocarditis are often exquisitely painful, and suppuration often occurs. The pain from loose cartilages in the knee joint is sudden and severe. The bony surfaces may grate in the joint affection of tabes and yet give no especial pain, and the same absence of pain is often noted in tuberculous joints. In Parrot's disease, scurvy, hemophilia, osteomyelitis, osteosarcoma and periostitis near the joints, pain is a more or less prominent symptom.

## TENDERNESS

We give this name to pain caused by pressure, although in many cases spontaneous pain of less degree is also present. The same caution must be used in estimating the value of tenderness as a symptom as is called for in the case of spontaneous pain, and the one may be simulated as readily as the other. Tenderness may be superficial, as seen in making pressure upon a boil, or deep, as in the case of an appendical abscess located behind the cecum. In the latter

case one must distinguish the hyperesthesia or increased sensitiveness of the skin, often noted over the inflamed part, from real superficial tenderness.

### THE HEAD

Tenderness of the scalp may be due to contusions or other injuries, periostitis, myalgia, neuralgia, migraine or other external conditions, or occur in association with disease within the skull—especially where the meninges are involved, as in gumma or other tumor, or abscess. It is best elicited by tapping gently over the area to be examined. The mastoid region should be tested in this manner if any suspicion of mastoiditis exists.

In disease of the face this sign is frequently found. The region over the antra and frontal sinuses is often tender to a light tap of the finger, and such tenderness should lead to a careful inquiry as to nasal discharge or other symptoms of sinus disease. Tenderness at the tragus is found in otitis media. The points of emergence of the branches of the trifacial nerve from the bony canals are tender in neuralgia of that nerve. The finding of a tooth tender to pressure or a light blow may throw light on the cause of the neuralgic pain. Hyperesthesia of the face and neck is common in neuralgia. In parotitis, suppurative adenitis, acute torticollis, neuritis of the vagus, tuberculosis or other acute disease of the larynx, caries of bone, etc., tenderness of various portions of the neck is found. The rigid neck muscles of meningitis are often tender. In herpes zoster facialis and cervicalis, and inflammation of the tear duct, tenderness is often extreme.

### THE CHEST

In all cases presenting pain in the trunk it is well to percuss the spine forcibly, and to ask the patient, if up and about, to rise on the toes and drop sharply on the heels; or the physician may make sudden pressure on the top of the head when the neck is in line with the trunk, to determine if tenderness exists in the spinal region. Meningeal, periosteal, arthritic, or bony disease is likely to be de-

tected at once by one of these procedures. We must differentiate from these more serious troubles neurasthenia, hysteria and lumbago, which also cause tenderness. Mannkopf described the marked increase in the rapidity of the pulse caused by pressure upon tender points in the spine.

Diseases within the chest, especially those involving the pleura or pericardium, or eroding the bony wall, give rise to tenderness upon pressure, as tuberculosis, pleurisy, pneumonia, pericarditis, subphrenic abscess, empyema, aneurism, mediastinal tumor, enlarged tracheobronchial glands, etc. Percussion over the sternum is painful in syphilitic disease of the periosteum, and should excite suspicion of lues unless otherwise explained. It is not infrequent from tuberculous disease of the sternum or ribs, in typhoidal periostitis, caries and arthritis deformans.

The tender points of Valleix in intercostal neuralgia are found generally under the left scapula, the left axilla and under the region of the apex-beat of the heart. The tender breasts of the menstruating period are common, and tenderness is found also in hysteria, early pregnancy, lactation, abscess, and in various tumors of the breast.

In herpes zoster the tender area may sometimes be detected before the appearance of the eruption, while anesthesia often follows the disease. Tenderness may be found in a narrow zone about the chest in a limited myelitis, or other local cord disease. In pressure neuritis involving the intercostal nerves, as especially in aneurism of the descending aorta, marked tenderness is often found.

## THE ABDOMEN

Most important of all is abdominal tenderness. When general it is symptomatic of a diffuse inflammation of the peritoneum or some of the abdominal organs, as in severe typhoid and in peritonitis. When local it frequently implies a limited peritonitis over some single inflamed organ, as in appendicitis, gastric ulcer, or cholecystitis. Tenderness along the rib margin at the attachment of the diaphragm is often found in severe cough, especially when the ab-

domen is lax, as after delivery. It is found in the wall of the abdomen in neuralgia, herpes zoster, in myalgia, and in neuritis.

Tenderness in the epigastrium is probably most frequently found in gastric ulcer and is one of the important diagnostic points in this disease. When well marked it signifies that the local ulceration has extended to the peritoneal coat, and when exquisite, perforation is probably present or imminent. Since gastric ulcer often obstructs the pylorus, and therefore causes dilation of the stomach, the ulcer may be much lower than the normal size and position of the organ would permit.

Tenderness also exists from gastritis, in acute dilatation, and in cancer of the stomach. Slightly to the right of the center we find the moderate tenderness of chronic inflammatory conditions in the gall-bladder, or the acute form found in suppurative and gangrenous cholecystitis, and in acute distention of the gall-bladder and ducts. After an attack of gall-stone colic, the tenderness may be quite marked for several days. In typhoid cholecystitis this sign is the best guide to the diagnosis.

Acute pancreatic disease causes great tenderness in the center of the epigastrium or slightly to the left. Pressure immediately under the tip of the sternum is painful in pericarditis with effusion, and upwards under the ribs, especially on the left side, in diaphragmatic pleurisy.

In the right hypochondrium we find tenderness in abscess and cancer of the liver, perihepatitis, acute yellow atrophy, acute hepatitis, catarrhal jaundice, cholangitis, or other acute disease involving the liver. If sharply localized it may point to a limited peritonitis over a cancerous nodule, or over an abscess. In ascending infection from the appendix the tenderness may be traced downward, at least in the early stages. Acute ulcerative conditions in the bowel, as in duodenal ulcer, and cancerous or other growths, cause tenderness here as elsewhere. The kidney may be the cause of the same sign if inflamed, as in pyelitis, calculous disease, malignant growth, perirenal abscess, etc. If the ureter be blocked in intermittent hydronephrosis, the swollen kidney is often especially tender.

In the left hypochondrium we must think especially of acute

congestion of the spleen, perisplenitis, abscess, cancer, tuberculosis, infarct, etc. The kidney and the colon must be considered, as upon the other side. Acute tenderness upon both sides may be found in influenza and in the associated perihepatitis and perisplenitis of leukemia.

In the umbilical region tenderness has less special significance than elsewhere, but is found in peritonitis, in the distended abdomen of typhoid, and other diffuse inflammatory processes.

Over the bladder region we have acute tenderness, when that organ is overdistended, as often seen in typhoid, and when inflamed, as in cystitis. Uterine diseases also frequently present this sign.

In the right iliac region we think first of typhoid and appendicitis as causes of the tenderness. In the latter disease acute tenderness presumably signifies that the appendix lies well toward the front, under McBurney's point; a lesser degree, that it lies further back from the abdominal wall, or that the inflammation is only moderately severe; if far to the right, or found rather toward the loin, that the affected organ is post-cecal in location. If little or no tenderness exists, while the symptoms point to a sharp attack, the tenderness may often be detected high upward to the right by rectal examination. This should never be neglected under these circumstances.

In renal calculus, hydro- and pyonephrosis, and other diseases of the kidney, and in cancer, obstruction, hernia and other affections involving the bowel, tenderness is often perceived in this region, and in the corresponding one on the left. Low down, toward the small pelvis, diseases of the tube or ovary, upon either side, give rise to tenderness, very acute in suppurative salpingitis and extra-uterine pregnancy.

In the left iliac region we have tenderness, as noted above, in conditions also found upon the right side. Less especial diagnostic import attaches to the sign here than in any other quadrant of the abdomen. A displaced appendix or cecum is not very infrequent here. Rarely diverticulitis is the cause of the sign considered. Dysentery and mucous colitis may also present it.

In the extremities tenderness may be due to trauma, neuralgia, neuritis, especially multiple neuritis, thrombosis of veins, or to almost any disease of bone or periosteum, joint or muscle. The infectious diseases of the joints, the hysterical joint, and the myositis of trichiniasis should be especially mentioned. Tenderness in the calf in continued fevers often precedes the development of venous thrombosis. In alcoholic and especially in arsenical neuritis the muscular tenderness is often exquisite. It may be seen in the tissues of the hands or feet as well as in the muscles of the arms or legs. The tender-toes of typhoid represent the effect of a very limited peripheral neuritis.

## SECTION III

### PARESTHESIA, TREMOR, VERTIGO, COMA, ETC.

#### **PARESTHESIA**

WHEN normal sensibility is distorted we have various forms of paresthesia as a result. The sensory nerves are irritated by some abnormal influence, heat, cold, some poison to the tissues, either generated within the body, as in gout, or introduced from without, as in the case of drugs, or applied to the skin or mucous membranes, as in ivy poisoning. As a result we may have itching, burning, numbness, tingling, formication, throbbing, feeling of constriction, coldness, heat, and various sensations described in most various ways by different patients, more especially neurotic females. All causes which lower the general resistance predispose to the development of these morbid phenomena. We have no means aside from the patient's word and manner for estimating the genuineness or severity of the troubles complained of. Tired and neurasthenic brain workers complain of various, oftentimes almost fantastic, sensations in the head, and these are more distressing if dwelt upon.

Perhaps numbness is the most common of these phenomena. Slight pressure, retardation of the venous circulation, or other cause slightly impairing the nutrition of the nerve, is sufficient to produce it. The immersion of the hands in hot water, as in women in certain employments, and occasionally in the use of hot water therapeutically, may cause the slight numbness and tingling in the hands and wrists described as acroparesthesia. Aconite and bromids are amongst the drugs which may produce numbness. Neuralgia, neuritis, herpes zoster, certain diseases of the brain and spinal cord, hysteria, neurasthenia, and myxedema may have numbness as a symptom. Nervous women at the menopause may complain of it on awakening. Tingling sensations are the ones most frequently associated with numbness. Meralgia, as occurring in the legs of men chiefly, and



only upon standing or walking, is often more troublesome because of numbness and anesthesia than because of pain.

Itching is perhaps the most distressing of the parasthesiæ. It is often associated with burning sensations, especially when the skin is congested, as in certain skin eruptions. Most important from the diagnostic standpoint are the forms associated with the elimination of certain poisons through the skin—morphin, biliary matter in jaundice, and urinary irritants in uremia and diabetes, in the latter especially in the genital region. Senile pruritus and that of gouty and lithemic patients deserve mention.

The peculiar sensation allied to itching, complained of by cocaine habitués, and described as a feeling as of worms burrowing under the skin, should be mentioned.

Sensations of heat and cold are common. If the feeling of heat becomes painful we speak of it as *causalgia*. The hot flushes of the menopause are extremely common. The chill of onset of acute disease is distressing, but the internal temperature is elevated. Myxedematous patients complain of cold, and in those whose resistance is reduced, as in anemia, tuberculosis, and certain nervous diseases, coldness may be complained of when none actually exists in the part, or when the local temperature is really below normal, as in the cold feet of feeble invalids.

Feelings of great weakness, of sudden debility, faintness, of oppression in the chest, of great weight upon the chest, as in nightmare, and many similar phenomena are complained of by neurotic patients. The possibility of a definite organic cause should never be forgotten.

### TREMOR

In many diseases we have a fine vibratory movement of certain parts due to alternate contraction and relaxation of the muscles controlling them. In alcoholism, and from over use of tea, coffee, and tobacco, in lead and mercurial poisoning, neurasthenia, under great excitement, or on account of cold, tremor is often seen.

That from alcohol may be temporarily lessened by a return to alcoholic drinks. It reaches its acme in *delirium tremens*, and chiefly affects the hands and lips.

Senile tremor has a rate of about five (5) oscillations per second, affects the hands most frequently, but also the head and lower jaw in certain cases. It is worse upon intentional movements, but may finally be noted in rest.

The most striking variety of tremor is that of paralysis agitans. It has a rate of three to six per second, is slightly under the control of the will, continues during rest, but generally disappears during sleep. The head generally escapes. The movement of the forefinger upon the thumb gives rise to the name "pill-rolling tremor."

In multiple sclerosis we see the variety of tremor characterized as intentional, absent in rest, but developing with greater and greater oscillations as voluntary movements are attempted. In earlier stages the rate is about eight per second, but lessens as the disease advances. It is seen in younger individuals than the two last described varieties.

A fine tremor is one of the chief signs of exophthalmic goiter, the rate being about eight per second. In typhoid and other low fevers, tremor is marked, affecting the lips and tongue especially. In tuberculous meningitis and certain tumors of the brain it is also seen, but it has no especial diagnostic importance. In certain families hereditary tremor develops in middle life, affecting chiefly the hands, although the legs and head are often later involved. It continues till death.

### FIBRILLATION

Individual groups of muscle fibers contract in a sluggish wavelike manner in certain diseases, and without effect in causing motion in the part involved. Fibrillation is of especial significance in those chronic degenerative nerve affections, in which the motor cells in the anterior horns are involved, as typically in progressive spinal muscular atrophy and bulbar paralysis. The fibrillation may often be brought out more distinctly by tapping the muscle under examination if it does not occur spontaneously.

### VERTIGO

Vertigo, known also as dizziness or giddiness, is due to some disease of that portion of the nervous system which governs the re-

lationship between the body and the surrounding objects. Lesions in the terminal filaments in the semicircular canals, in the vestibular portion of the auditory nerve, the pons, cerebellum or crura cerebri, may be responsible for this symptom. Nausea, vomiting, faintness, and great distress accompany severe vertigo. Consciousness is never completely abolished, for it is essential to the perception of vertigo.

Vertigo is due to:

(1) Mechanical causes, such as rapid revolutions of the body, swinging, unusual positions, and rapidly changing positions, the latter especially in car-sickness and seasickness.

(2) Toxemic conditions. In acute indigestion it is very common, especially so in meat-poisoning, often associated with headache. In dilated stomach with anacidity and fermentation of food, vertigo is especially common. Many patients apply for relief for the latter symptom without being aware of serious indigestion. Gastric vertigo is not accompanied by deafness, tinnitus, nor pain in the ear, unless by accident. In poisoning by alcohol and tobacco it is familiar, and many narcotic poisons also produce it. Lithemic individuals and those subject to bilious attacks commonly complain of vertigo on arising in the morning.

(3) Circulatory disturbances. In anemia of the brain, as in faintness, or hyperemia, as after inhaling nitrites, vertigo is often seen. The vertigo of cerebral arteriosclerosis, a very common and distressing symptom, and that of aortic regurgitation and myocarditis, are due to the resultant anemia. Tinnitus aurium and increased vascular pressure may be associated.

(4) In organic brain disease, more especially in tumor, in brain syphilis and in meningitis. In mastoid disease it is a symptom ominous of extension of the inflammation to the brain. Ear symptoms are not present as a rule in cerebellar vertigo, while staggering gait is characteristic. Vertigo from brain disease and from changes of intracerebral pressure arises from the associated cerebellar disturbance.

(5) Local irritation of the external auditory meatus, as from cerumen, or syringing out the ear, may cause vertigo.

(6) Vertigo occurs in labyrinthine disease associated with nys-

tagmus—true auditory vertigo. This is the type in Ménière's disease. The attack is sudden and is usually followed by deafness.

(7) In cerebral concussion, sunstroke, neurasthenia, epilepsy, and other neuropathic disturbances, vertigo may be present. Psychical disturbances may produce it, as in the dizziness experienced when in high places.

(8) It is common in disturbances of the vision, since the cerebellum, the organ of equilibrium, receives a part of its impressions directly from the eyes. The attack may cease upon closing the lids. In Japan the so-called "paralyzing vertigo" is described, and this has also been described in epidemic form in Switzerland. It is a disease of warm months, and is accompanied by general weakness or even general paralysis, without loss of consciousness.

In locomotor ataxia, when the general sensibility and muscular sense are greatly affected, vertigo may accompany the staggering gait. In this disease we most often see laryngeal vertigo, or laryngeal epilepsy, a sudden suffocative strangling attack, with cough and dyspnea, and even asphyxia and loss of consciousness for a brief interval. Convulsive movements may occur. These attacks may also be seen in asthmatics and those affected with other lung troubles.

## CONVULSIONS AND SPASMS

Although custom permits much latitude in the use of these two terms, the best usage defines a convulsion as a paroxysm of violent and involuntary general muscular contractions, often accompanied by unconsciousness, and limits the use of the term spasm to a similar process of a local nature, often without loss of consciousness.

A. tonic convulsion presents continuous muscular contraction, brief, as in the beginning of an epileptic attack, or of long duration, as in lockjaw. A clonic convulsion is characterized by alternating contraction and relaxation of muscles, as in the midst of the epileptic paroxysm. In the tonic convulsions of tetanus, of strychnin poisoning, etc., consciousness is preserved, but is generally lost in the clonic type, as in eclampsia. In the tonic type there is little movement of the body and limbs, while the reverse holds in the clonic con-

vulsion. Spasm generally affects a single muscle or group of muscles. It is termed cramp when tonic in character and painful and generally affecting a single muscle or a group working together, as in the calf of the leg.

The term Jacksonian epilepsy refers to the limited convulsive movements arising from cortical irritation—one of the most valuable indications in the localization of cerebral disease. Tetany is characterized by peculiar local spasms affecting chiefly the hands and feet. The spastic movements in the different varieties of chorea are limited to single muscles or muscular groups. General convulsions with unconsciousness are due to some irritant carried to the brain in the blood stream, as in uremic and alcohol convulsions, or to some direct or indirect irritation of the organ, as the pressure of a growth, or a reflex from a diseased organ elsewhere.

Local irritation may be direct, as from a hemorrhage or fracture of the skull, tumor, cyst, etc., affecting the motor centers, or indirect, as by transmitted pressure, interference with the circulation, etc.

Convulsions from general irritation of the brain may be from unknown cause, as in epilepsy; from drugs or other poisons from without, such as lead, alcohol, strychnin, etc.; from those generated within the body, as in uremia and asphyxia; from the poisons of an infectious disease, as in children at the onset of scarlet fever, or in the course of chronic diseases, especially rickets; from anemia of the brain, as after hemorrhage; from congestion of the brain, as in hanging; from the cortical irritation accompanying certain diseases of the brain, such as the inflammation of meningitis, or the degenerative changes of parietic dementia.

Reflex convulsions arise from some powerful influence operating upon the brain from a distance, as in the severe pain of renal colic, or after violent injuries. Sensitive children may have convulsions from the irritation of teething or of indigestible food. The occurrence of convulsions is of serious moment in children, since lesser causes will excite them if the habit become established. The convulsions of hysteria are less severe than those we have been considering, excepting in the case of hysterio-epilepsy, in which more violent movements and unconsciousness are often present.

## ABNORMAL PSYCHIC CONDITIONS

Abnormal psychic conditions are so common in other than purely mental diseases that every physician must pay some attention to them.

### INTELLIGENCE AND MEMORY

The variations in degree in the development of these faculties are so great, according to the race, age, native mental ability, opportunities for education and general environment, that nothing but a wide experience with people in all walks of life will guide the physician in determining whether they are normal for the particular individual under consideration. A marked change from a previously known condition is of great significance.

Idiocy, imbecility, and dementia are likely to attract the attention of the physician as the first question is asked, if the filthy, unkempt or inattentive manner of the patient has not already made apparent some form of mental deficiency. The milder grades of lack of intelligence, such as dullness, stupidity and congenital weak-mindedness, become apparent upon further examination. These various grades of mental disability may depend upon congenital structural defects, as in idiots, or upon damage inflicted upon the normal brain at birth, as in infantile hemiplegia, or upon faulty development, as in cretinism. Memory can scarcely be tested for lack of a proper degree of intelligence in many of the classes of defectives considered.

The development of mental trouble in those previously of normal intelligence is an extremely frequent phenomenon, and arises from the most varied causes. Simple temporary exhaustion may so weaken the brain for the time that the memory becomes defective, and all the mental faculties less alert than usual. The failure in cerebral nutrition in the arteriosclerosis of advancing years often renders the memory, especially for recent events, inaccurate and even wholly defective. Pronounced chronic anemia, especially that of hook-worm disease, and the pernicious form, render the patient dull, stupid, and apathetic. Chronic diseases which impair nutrition, as cancer, heart disease, cirrhosis of the liver, starvation, etc., are often accompanied by progressive failure in mental power.

Coarse cerebral damage, as tumor, hemorrhage, thrombosis, syphilis, the cortical changes of parietic dementia, or the results of traumatism, gradually undermines the intelligence, even though consciousness be not lost. The development of myxedema, cachexia strumipriva and epilepsy may act in a similar manner. The intoxication of acute infectious diseases, notably typhoid fever, impairs the intelligence in various degrees until coma is reached. Deficient mentality may be apparently present when aphasia, agraphia, or other lesion of the mechanism of expression is the chief or even the only factor indicating it.

Exaltation and depression are, in minor degree, normal mental phenomena, and may even be felt with changes in the barometer. The administration of alcohol, ether, and other drugs, congestive conditions of the brain, as in the delirium of acute fevers, and certain mental diseases, notably acute mania, are associated with mental exaltation. Depression is very common in those exhausted by chronic disease, and in neurasthenics, hypochondriacs and hysterics. The periods of depression associated with chronic gastric and hepatic disease and with the menopause are well known to the laity. Painful diseases and exhausting streptococcic infection are especially prone to present this symptom.

Irritability is so common in otherwise normal individuals that it must not be looked upon as pathologic unless it passes reasonable bounds. High-strung, energetic, and intellectual men are often notably irritable, and especially after hard and prolonged mental labor, more particularly if it has been accomplished under stimulation by tea or coffee. Any unusual expenditure of nervous force is likely to leave the subject not only irritable but depressed, as in the case of the clergyman's "blue Monday." Nearly all invalids are oversensitive and irritable to some degree, but those with the uric acid diathesis and gout are notably so.

### DISEASES OF SLEEP

In general terms sleep becomes less sound as we advance in age, and less hours of rest are necessary. Few individuals are able to

remain in health with less than five or six hours of sleep, and these are likely to be past middle age. In the main working period of life, seven to eight hours is the average demanded, while children demand more and more as we approach infancy. Men usually require less sleep than women. There is the greatest diversity in the sleep habits of different individuals. One is prostrated over the loss of a part of one night's sleep, and another, like Napoleon or Edison, may perform Herculean labor with but four hours rest in the twenty-four. Manual labor requires more sleep than mental, but the quality of mental work falls off more rapidly than that of manual labor if the hours of sleep be too much curtailed.

**Insomnia.**—By insomnia we mean the inability to sleep normally. As a functional disorder we see it in patients of nervous temperament, and especially those who use the brain rather than the muscular system. Irregular hours of sleep, the untimely or excessive use of tea and coffee, the use of alcohol and tobacco immoderately by certain persons, over-excitement, and that over-sensitiveness of the nervous mechanism seen in neurasthenics, predispose to sleeplessness. In mania and other types of insanity, especially paresis, in delirium tremens, in the initial stages of many of the acute infectious diseases, in many diseases accompanied by increased intracranial pressure, exophthalmic goiter, and in most diseases accompanied by continuous pain or other form of irritation, it is a prominent symptom.

In anemia of the brain from arteriosclerosis or from cardiac disease, it is often distressing. In the aged it is frequently present as a mild but slightly troublesome symptom. In a child it should call for careful search for some serious organic cause. One of the chief reasons for the lack of endurance of the neurasthenic is the lack of rest for his nervous system at night, because of sleeplessness or that light slumber in which he still suffers mentally from the worries of the day.

**Somnolence.**—This occurs in various organic cerebral diseases, notably in cerebral syphilis, dementia, and certain types of arteriosclerosis. Various toxic substances may cause it, as those found in diabetes, and alcohol, opium and other drugs, and the poisons of



uremia. Insufficient supply of blood to the brain, as in anemia of heart disease; the toxins of the *Trypanosoma Gambiense* in African sleeping-sickness; obesity, as in the case of Dicken's fat boy; myxedema and various forms of insanity have drowsiness as a prominent symptom. Increase or decrease of blood supply, under certain conditions, may be associated with either wakefulness or drowsiness, and neither is due exclusively to one cause.

**Narcolepsy.**—Narcolepsy is abnormal sleep of unknown cause, terminating in coma and death in some instances.

**Dreams.**—Dreams are due to the action of certain cortical centers when others are inactive because of sleep. An overloaded stomach, distended bladder, an uncomfortable position in bed, or some other distressing sensory impression furnishes the basis. Nightmare in adults and pavor nocturnus in young children are vivid dreams with sensations of oppression in the chest, of horror, and inability to escape some impending catastrophe. Both proceed from indigestion in most cases.

**Somnambulism.**—In sleep walking or somnambulism, and sleep talking or somniloquy certain brain centers remain active during sleep, and conversation, walking about, unlocking doors and other feats possible to the waking under usual conditions are performed readily, and generally with immunity from the dangers one might expect. These conditions occur in neurotic individuals, as dreams occur in others, often from indigestion, anxiety, or similar cause.

**Disturbances of Consciousness.**—Under the heading somnolence we have discussed the first step toward loss of consciousness. Stupor designates a greater degree of loss, the subject being capable of being aroused at least partially by vigorous effort. In coma he is incapable of being aroused, and stertorous respiration and general relaxation are present in some degree.

## COMA

The term coma vigil designates the condition in which the patient lies unconscious with muttering delirium and carphologia, but with the eyes open. It is seen in typhoid and some other exhaustive acute diseases, and is of extremely grave omen.

The significance of coma is often difficult to determine, and especially so when we have no history of the case to guide us. The different types considered are arranged approximately in the order of their frequency in general practice, although no exact order could possibly be established.

**1. Coma Preceding Death.**—This is generally easily recognized by the associated signs and symptoms.

**2. Coma from Alcoholism.**—Convulsions are occasionally seen. The known habits of the individual, odor of breath, appearance of skin of the face (congestion, acne), flushed face, or pale at times, or even cyanotic, moist skin, oftentimes cool from increased radiation of heat, frequent, full pulse, or later, the small pulse of exhaustion, equal pupils, normal in size or dilated, a frequently lowered bodily temperature and stertorous respiration will guide us to the diagnosis.

**3. Coma from Anesthetics.**—The odor and general surroundings should direct us in the rare cases when the explanation is not immediately apparent.

**4. Coma from Uremia.**—Uremic odor of breath and sweat, edema, hypertrophy of heart, and increased blood tension in many cases, normal and equal pupils, convulsions, albuminuric retinitis and presence of albumen and casts in the urine suffice for the diagnosis in most instances. It is possible that the uremic patient may also have had an apoplectic stroke or be intoxicated. Since hemiplegia may be purely uremic in origin a reservation should be made as to the character of a stroke of apoplexy developing in a patient with nephritis.

**5. Coma from Cerebral Hemorrhage, Syphilis, and Other Organic Brain Diseases.**—If a history can be obtained we may determine quite closely in most cases the cause of the coma of brain disease. That of syphilitic origin is likely to develop gradually and often without definite localizing paralytic signs. That from cerebral thrombosis is preceded by numbness, tingling and formication, and comes on gradually. The coma of cerebral hemorrhage and cerebral embolism is often instantaneous in origin, and high arterial tension, or signs of valvular disease or other source of embolism, point toward the one or the other. The face may be flushed, cyanotic, or pale; the respira-

tions are slow and often stertorous, the pupils are often unequal and do not respond to light; and the signs of hemiplegia are present—flapping paralyzed cheek, drooping angle of the mouth, conjugate deviation of the eyes, abnormal flaccidity and loss of skin reflexes, and the preliminary decrease and reactionary increase of temperature of the affected side. A gradually increasing coma is present in ingravescient apoplexy, the hemorrhage in this type bringing about a full hemiplegia only after some hours, presumably from a very slowly increasing pressure upon the portions of the brain exposed.

**6. Coma of Typhoid, Malaria and Other Acute Infectious Diseases.—**

The general character of the febrile diseases suffices for the diagnosis in most cases. The malarial plasmodium must be sought for if a pernicious malarial paroxysm be suggested.

**7. Coma of Traumatic Origin.—**Injuries of the skull should be sought for with great care if no history is obtainable. No ambulance case should be passed upon without this precaution, the head being shaved if necessary. Escape of blood or cerebral fluid from the ears should be looked for.

**8. Coma from Narcotic Poisoning: Opium and its Derivatives, Chloral and Other Narcotics.—**The slow pulse and respiration, cyanotic face, warm and dry, and later moist skin, and contracted pupils, point toward an opiate. In large cities many patients are seen, unconscious from the “knock-out drops” used by criminals—a solution of about forty grains of chloral, generally given in beer or other liquor. The history may be needed to clear up the diagnosis as to the particular drug used.

**9. Coma from Asphyxia, Drowning, Etc.—**In asphyxia, drowning, and other suffocative accidents the history and surroundings must guide us.

**10. Coma from Epilepsy and in Association with Convulsions of Other Type.—**In epilepsy, puerperal convulsions, convulsions of infectious diseases in children, and infantile eclampsia, coma often alternates with convulsions, as is often the case in uremia. The scars of the bitten tongue may be present.

**11. Hysterical Coma.—**The preliminary manifestations of hysteria and the general aspects of the case are likely to give a clue to the

diagnosis. The coma is less profound than in most of the diseases we consider, and the patient may generally be partially roused by pressure upon the supra-orbital nerve, or other painful impression.

The absence of unequal and irresponsive pupils, the turning up of the eyeballs, resistance to opening of the lids, the good color and normal or merely excited heart and lung action, all point toward the diagnosis of hysteria. Many cases, however, of serious brain disease have associated hysterical symptoms, and search for any signs of such trouble should be made no matter how clear the diagnosis may seem.

**12. Coma from Syncope.**—Unconsciousness from syncope is readily recognized by the pallor, weak or absent pulse, and the knowledge of some cause, psychic, as at the sight of blood, or, as in the case of a weak heart, physical, as when a patient weakened by long illness sits up for the first time. Simple syncope is usually of very short duration if the patient be placed in the horizontal position and the usual remedies be applied.

**13. Coma from Diabetes.**—Diabetic coma is often preceded by drowsiness, the odor of acetone may be detected in the breath or even in the air of the room; sugar, acetone, and diacetic acid are found in the urine and frequently the red flush of the cheeks, eczema about the genitals, boils, dry skin, gangrene or other more or less characteristic features of diabetes may be seen. Dyspnea is very striking in certain cases.

**14. Coma from Gas Poisoning, Carbon Monoxid, and Carbon Dioxid.**—The increasing use of water gas containing much CO has changed the picture of gas poisoning, for the red color of the blood saturated with this compound is seen instead of the cyanotic hue of CO<sup>2</sup>. The history commonly shows poisoning by illuminating gas or by coal or charcoal fire, or exposure, in mines, wells or pits, to the poisonous gas.

**15. Coma of Sepsis, as in Septicemia, Pyemia, Acute Yellow Atrophy of the Liver, Etc.**—Such unconsciousness is generally easily recognized by the associated clinical features.

**16. Coma from Sunstroke and Heat Prostration.**—The unconsciousness of sunstroke is often preceded by convulsions. The history, the

extreme heat of the day or of the place where the attack occurred, the heat of the skin, the fever, reaching to from 108 to 114 degrees, render the diagnosis easy in most cases. In heat prostration the fever may be absent and the skin cool, but the history makes the case clear.

The danger that coma from a fractured skull or apoplexy may be mistaken, for example, for that of uremia or alcoholism should always be borne in mind in these cases. Two causes may coöperate. Thus I have seen to-day an epileptic with uremic convulsions and coma.

### DELIRIUM

This occurs under a great variety of conditions. The patient is excited, even maniacal, restless and incoherent (active delirium), or he is quiet and only mildly restless, inattentive to his surroundings and muttering incoherently (low muttering delirium). All gradations are found between these types.

Children and neurotic individuals are especially subject to delirium. It may appear suddenly as at the beginning of an acute infectious disease or develop gradually, showing only mildly at certain hours, especially at night. Somnolence, coma or convulsions may appear.

Active or maniacal delirium is especially common in acute mania, acute inflammatory disease of the brain, and acute infectious disease, especially sepsis and pneumonia. Muttering delirium is especially characteristic of typhoid and other low fevers, after the acute stage of onset is passed and nervous exhaustion begins. A mild delirium at night in patients recovering from any exhausting disease is of less significance than the form seen during the height of illness, which is to be regarded as a grave symptom.

In uremia and in poisoning by opium, hyosciamus, belladonna, cannabis indica and alcohol, delirium may be present. After an epileptic convulsion maniacal delirium may occur. In conditions of grave inanition, whether they arise from simple starvation or from malignant or other disease of the digestive tract, delirium is often present.

Delirium tremens is the name given to that form seen after the chronic use of alcohol, and developing either after a debauch or after the alcohol is suddenly cut off. There are present hallucinations, which constitute the most characteristic feature of the disease. The patient imagines that snakes, insects and other small animals are crawling about the room or upon the bed. The restlessness, sleeplessness and tremor are almost as characteristic as the hallucinations. A somewhat similar condition is sometimes seen after the long use of other drugs, especially of chloral and morphin. Delirium may be feigned, but it is usually easily detected. The picking at the bed clothes, carphologia, seen most typically in the low delirium of typhoid fever, and generally of fatal significance, depends upon the hallucinations of the disease.

### **DELUSIONS, ILLUSIONS, HALLUCINATIONS**

In insane patients and in others temporarily ill, especially those having delirium, and in hysteria and other functional nervous diseases, a group of disorders of consciousness may occur which demand consideration.

A delusion is a belief without foundation, and often absurdly ridiculous, such as that the subject is the Emperor Napoleon, or the richest man in the world. Such delusions of grandeur are often seen in parietic dementia. The subject cannot be reasoned out of his false belief.

An hallucination is "a false perception of the senses for which there is no external cause," and any of the senses may be affected. The patient who believes he hears a voice when none exists suffers from an hallucination.

An illusion differs from an hallucination in that some actual basis really exists for the false perception. A voice of an attendant is actually heard, but the subject believes it to be the voice of an angel, for instance. A patient may or may not recognize the falsity of illusions and hallucinations.

Obsessions of doubt, of fear, or of the most various kinds, the impulse to touch certain objects, or to perform certain acts, and the

many fixed ideas noted among the insane are more properly discussed in text books upon mental diseases.

## **SIGNS AND SYMPTOMS PERTAINING TO THE URINARY AND GENERATIVE ORGANS**

### **MICTURITION**

The use of the ureteral catheter shows that urine is ejected from the pelvis of the kidney intermittently. It collects in the bladder and is evacuated in health some five or six times in the twenty-four hours. The amount averages about 1500 c.c. in adults, slightly less in the female sex. Children pass more in proportion to their weight than adults, because of their more active metabolism.

By oliguria we mean lessened excretion of urine. Anuria signifies its complete suppression. Polyuria designates an abnormal increase in the amount, as in diabetes, after hysterical attacks and in certain types of chronic nephritis.

**Oliguria.**—Oliguria may depend upon lessened ingestion of fluids, upon disease of the kidneys preventing their proper function, or upon excessive loss of bodily fluids, as in cholera and other diarrheal diseases. It becomes more dangerous as it approaches anuria.

The urine may be suppressed from disease of the kidneys preventing its excretion, such as acute nephritis and acute congestion, possibly supervening upon a chronic nephritis; suppurative diseases in or about the kidneys, or interference with the blood supply by aneurism, thrombosis or embolism.

The oliguria of cholera and other diarrheal diseases may progress to complete anuria. In shock from various abdominal diseases or operations, and after severe hemorrhage, anuria from lowered blood pressure occurs.

As the result of blocking of the ureter by stone when but one kidney exists, or by reflex suppression in the other kidney when one is so blocked, or by blocking of both ureters simultaneously, after the mistaken removal of the only functioning kidney, and in hysteria and sunstroke, complete suppression may be seen. The patient

dies in less than a week in most instances, sometimes without the usual uremic symptoms.

Since suppression is not generally absolute, the normal findings in any urine that may possibly be obtained suffice to establish some mechanical trouble and not disease of the kidneys as the cause of the suppression. In the latter case, the presence of blood, albumin, casts, pus cells, bacteria, etc., will point toward the proper diagnosis.

**Retention of Urine.**—This is distinguished from suppression by the finding of urine in the bladder, though it cannot be passed voluntarily. The retention may result from mechanical causes, such as pressure upon the urethra in labor, an enlarged prostate, a foreign body in the urethra, a thread tied about the penis, as is occasionally seen in boys, stricture of the urethra, or urethritis. Reflex spasm of the vesical sphincter may cause retention, as may be seen during the passage of a renal calculus through the ureter.

In functional nervous diseases, notably in hysteria, in conditions affecting the brain, as in the coma of typhoid, or the cord, as in myelitis, retention occurs, oftentimes alternating with incontinence. In belladonna poisoning retention is common. Should it occur in an acute abdominal disease, it establishes a fair presumption that the peritoneum over the bladder has become involved in a peritonitis, as is most frequently seen in appendicitis. The over-distention of the bladder in elderly men often causes it to lose its power of contraction for a time.

**Incontinence of Urine.**—In most cases this is of nervous origin. If the brain, from injury or disease, loses its power of control, as in shock, in the various types of coma, and in the idiotic and the demented, incontinence may occur. In injury or disease of the cord, notably in myelitis and locomotor ataxia, and, perhaps most frequently, in hospital practice, in those cases of transverse myelitis from spinal injury, the dribbling urine is a distressing feature.

If the vesical center in the sacral region of the cord be incapacitated by disease, we have a paralysis of both detrusor and sphincter muscles of the bladder, with retention and overflow of urine—the incontinence of over-distention. It is safer to assume that dribbling urine indicates retention with overflow until the contrary is proven,



because of the danger that this condition will lead to cystitis, pyelitis and fatal damage to the secreting tissues of the kidney.

Incontinence also occurs as the result of increased reflex excitability of the bladder, as from a concentrated or irritating urine, cystitis, stone in the bladder, phymosis, seat-worms, or other cause of irritation. The nocturnal incontinence of neurotic children is generally due originally to the irritation of a too concentrated urine, the frequent repetition establishing a habit very hard to break up and even persisting after the original irritation has disappeared.

Women frequently suffer from incontinence as the result of injuries from parturition, and from various diseases of the pelvic organs. The sphincter gives way upon any slight increase in the intra-abdominal pressure, most commonly from coughing, sneezing or laughing. This is very frequent in pregnancy as the result of a simple relaxation of the sphincter without structural disease of the parts.

**Dysuria.**—Dysuria or painful micturition is best understood by describing separately the different factors which in various combinations cause the distress.

By tenesmus we mean the painful, spasmodic and almost continual efforts of the bladder to empty itself, even though it contain but a small amount of urine. Violent bearing-down pains and distressing burning of the urethra and perhaps rectal tenesmus are present.

Strangury is often used with much the same meaning but should properly indicate a condition with greater pain and spasm. It frequently results in the passage of but a few drops of urine, mucus or blood.

Dysuria may be caused by any condition of the urine making it irritating to the mucous membrane of the bladder and urethra. Too great concentration or acidity of the urine is an extremely common cause. After the absorption through the digestive tract, lungs or skin, of certain irritants, dysuria may result. Thus cantharides may be absorbed through the skin, turpentine through the lungs, and these and many other drugs through the stomach, and cause dysuria.

Over-indulgence in red pepper, so much used by certain national-

ities, or the free consumption of drinks flavored with ginger, etc., may even cause a temporary purulent discharge from the urethra, with much burning. Dysuria occurs in the vesical crises of tabes.

Inflammation of the urethra, as in gonorrhea or urethral chancre, or in connection with the eruption of a variolous pock, and of the bladder in the various forms of cystitis, in cancer, tuberculosis, enlarged prostate, and vesical calculus, may be the cause of severe dysuria. Severe inflammations in the region of the rectum,, by direct extension of the inflammatory process, or in a reflex manner, also give rise to this symptom.

In women dysuria often results from dysmenorrhea or from some disturbance in the walls of the bladder, or from disease of other parts. Thus ante flexion of the uterus, and other displacements to a less extent, pressure of tumors, prolapse of the bladder wall, because of ruptured perineum, and inflammatory adhesions of the wall to neighboring parts may act as causes.

**Slow Urination.**—This may result from obstruction in the urethra or insufficient expulsive power in the bladder, generally of nervous origin, but possibly because of inflammation extending to the walls from without. Careful physical examination will usually determine the cause.

**Frequent Urination.**—This occurs either because there is more urine to be passed, as in the polyuria of diabetes, or because the bladder is over sensitive and demands relief before becoming filled, as in cystitis, or because the bladder is smaller than normal, as after some types of inflammation. More common than any of these is the frequency resulting from purely nervous causes, as in neurotic individuals, and in normal ones under excitement.

The passage of blood, pus and various sediments in the urine is considered in the chapter devoted to the examination of that secretion.

## REPRODUCTIVE ORGANS

The multiform manifestations of the effects of venereal diseases, upon the original patient or upon others infected by him, are of such importance as to demand that we give the closest attention to the

investigation of the genitalia. Perhaps no other portion of the organism is so frequently the cause of neurotic, neurasthenic, hysterical and hypochondriacal symptoms. Serious psychological disturbances are by no means infrequent as the result of disease or the fear of disease of the genital organs. Males oftener than females consult the physician over some cause of mental trouble connected with the generative apparatus. The complaint is generally of nocturnal emissions, loss of semen at stool, the appearance of the (normal) viscid secretion at the meatus during sexual excitement, of slight varicocele, that one testicle is smaller or hangs lower than the other, or that he has contracted a venereal disease when none in fact is present.

Such patients are generally between 16 and 25 years of age, and have had their attention focused upon sexual matters from remorse over self-abuse or illicit intercourse, or from the reading of quack advertisements in the newspapers.

**Urethral Discharge.**—In either sex this may occur as the result of a simple urethritis or gonorrheal infection, a urethral chancre or other ulcer; in males, stricture, prostatitis and gleet should also be considered, and the more or less normal discharge, according to the circumstances, of the natural mucous secretion during sexual excitement, or of semen when the distended seminal vesicles are subjected to the combined pressure of a constipated-bowel movement, and the bearing down effort needed for its expulsion. The passage of semen under other conditions than these, and without the normal sensory phenomena of the sexual orgasm, is abnormal.

Nocturnal seminal emissions, several times in the month, occur in most healthy young men, and are physiological. Following sexual abuse of various kinds, and after gonorrhea, more frequent emissions are pathological and should be carefully investigated.

Phosphaturia, discharge of pus from prostatic disease, cystitis or chronic urethritis, must be eliminated. The depressed and distorted mental condition of sufferers or supposed sufferers from the various troubles mentioned furnishes the explanation of their resort to advertising quacks, and their frequent refusal to believe the statements of the physician that the condition explained is not of extreme gravity.

**Priapism.**—It is better to limit this term to the morbid condition of prolonged and frequent erections of the penis, without normal sexual desire, although it is often used to describe excessively frequent and severe but still normal erections.

Priapism results from various disturbances of the nervous system, notably myelitis, especially in the cervical and lumbar region, spinal meningitis, and lesions in the pons and cerebellum. In hydrophobia, tetanus, leukemia and alcoholism it may be present. It results most often from some irritation of the bladder or neighboring parts, as from the presence of a distended bladder, vesical calculus, prostatitis, gonorrhea, balanitis or phymosis. In male infants and in boys an excessively acid and hence irritating urine is a very common cause. In poisoning by cantharides and turpentine, and in association with inflamed hemorrhoids or other source of rectal irritation, it may be present.

**Varicocele.**—This is found in some five per cent. to ten per cent. of men. The spermatic veins are enlarged and tortuous, and cause aching and discomfort. Backache is frequently present. The condition of general relaxation and debility in which varicocele is common is often the cause of relaxation and elongation of the scrotum and spermatic cord, so that the testicles hang lower than normal, often to the annoyance of the patient.

**Onanism.**—Masturbation is so common in boys at some period of youth as to be almost devoid of pathological significance unless from excessive indulgence it produces definite symptoms. In that case they are the same as those of sexual excess. In the feeble-minded and certain insane persons onanism is a symptom of the nervous derangement rather than a cause. The conscience-stricken youth who attributes various more or less fantastic symptoms to his early indiscretions, and yet is normal in other directions, mental and physical, is a common figure in the consulting room.

**Impotence.**—This may be purely mechanical, from any cause preventing a normal erection. It may be due to drugs which depress the nervous mechanism upon which erection and ejaculation depend—notably the bromids, and, when used for a long period, opiates and alcoholics. A psychical form is not uncommon, due to fear, self-

distrust, mental abstraction, disgust or aversion toward some particular female. Exhaustion, from disease, age, starvation, diabetes and over indulgence in coitus, and certain organic nervous diseases, particularly locomotor ataxia and paresis in their stages of full development, are causes of impotence. Neurasthenic subjects with over-irritable nervous centers are frequently impotent because of the occurrence of premature ejaculation. Previous sexual irregularities are usually mentioned in the history of these patients.

### FEMALES

In the female sex we find more signs and symptoms dependent upon definite diseases of the sexual organs than in the male, and vastly fewer of the nervous psychical disturbances.

**Vaginal Discharges.**—A mild leucorrhea is so common, especially just before or after menstruation as to be scarcely pathological. A constant flow, especially if irritating or offensive, is of considerable diagnostic significance. Anemia, debility, severe work, especially in the upright posture, the irritation from uncleanness, from endometritic discharges, from gonorrhea, from uterine cancer or sloughing fibroid, and, in girls especially, from ascarides, are common causes. Innocent gonorrheal infection is so common in girl babies as to deserve especial mention.

Leucorrhea of gonorrheal origin is accompanied by much burning and swelling of the parts, and that from cancer and sloughing fibroid is likely to be blood-stained and offensive.

**Amenorrhea.**—Menstruation is normally absent during pregnancy, the earlier period of nursing and after the menopause, whether this be natural or induced by the removal of the ovaries, and is occasionally so from absence or lack of development of the female generative organs, or from imperforate hymen, which prevents the discharge of the secretion. The persistence of menstruation during the early months of pregnancy and its reappearance in the first one or two months of lactation are very common. On the other hand one patient of mine did not menstruate from just before the time of her marriage until after she had borne her ninth child, a period of seventeen years.

Amenorrhea is very common as the result, and not as the cause, as so many patients believe, of anemia, phthisis and many other exhaustive diseases. It is often caused by mental overstrain, worry and grief. In certain nervous affections, especially hysteria and melancholia, it is frequent. After change of social and climatic conditions, as in the newly married, or in those recently removed from the country to the city, especially if the change involve a sea voyage, amenorrhea is often noted.

After the destruction of the ovaries by disease, or their atrophy, as following the metastasis occasionally noted in parotitis, in consequence of the general physical deterioration in morphin habitués, and in the obese, absence of the menstrual flow is a frequent phenomenon.

**Dysmenorrhea.**—Difficult and painful menstruation may occur because of local disease of the sexual organs or on account of some general disorder. Under the first heading we may class the dysmenorrhea arising from flexions and other displacements of the uterus, narrow os, contraction from scars, disturbance of normal circulatory conditions by fibroid and other tumors, disease of ovaries and tubes, and other causes of mechanical disturbance. In membranous dysmenorrhea the diseased menstrual decidua is expelled with great pain, often leading to the suspicion of pregnancy unless the possibility of its occurrence is borne in mind.

In the second class we have nervous or neuralgic dysmenorrhea. No sufficient local cause is to be found, although a trivial displacement or other abnormality may be present. The disturbance is attributable to the anemic and ill-nourished general condition of the patient, or to the ill-balanced nervous organization. It is often seen in neurasthenic and hysterical girls, and even in women who have borne children. Complaint is made of unusual menstrual discomfort, nausea, vomiting and hyperesthesia over the lower abdomen, and there may be even convulsions, collapse, or coma.

Intermenstrual pain is the name applied to that variety of pain coming on at the middle of the intermenstrual period, often of great severity, and presumably due to some ovarian disease.

**Menorrhagia and Metrorrhagia.**—Abnormally profuse menstrual

flow constitutes menorrhagia, while hemorrhage from the uterus at other than the menstrual periods is termed metrorrhagia. Either may arise from local disease, and especially from those disorders, such as endometritis, fibroid and other tumors, polypi, displacements and subinvolution, which cause marked congestion of the pelvic organs. The uterine mucosa then bleeds more easily than under normal conditions. In those general diseases in which hemorrhages occur in various parts of the body, either form of uterine hemorrhage may be noted, as in hemorrhagic typhoid, hemophilia, scurvy, leukemia and the purpuric diseases. The menstrual flow is often increased in general febrile diseases, as in typhoid and in small-pox. In valvular disease of the heart, alcoholism, cirrhosis of the liver, nephritis and other chronic diseases, it is occasionally noted. A profuse flow should be accepted as a symptom of the menopause only after careful investigation as to the presence of endometritis, polyp, fibroid or cancer.

If metrorrhagia be present the possibility of miscarriage must be considered. If it occur after the menopause it should demand careful investigation because of the frequency of this flow as a symptom of cancer and submucous fibroid. Particularly offensive flow leads one to think of three possibilities—a septic miscarriage, a sloughing fibroid, and cancer.

## SECTION IV

### SYMPTOMS AND SIGNS PERTAINING TO THE DIGESTIVE SYSTEM

#### THE MOUTH

THE chief causes of complaint by patients, as regards the mouth, are of bad taste, of pain, and of dryness.

The first symptom is found in those types of indigestion associated with an over-loaded stomach or an inactive liver, in acute cases, and with chronic gastric and hepatic disease in chronic cases. The bad taste noted in an ordinary bilious attack is an example of the former, and that found in cancer of the stomach, dilated stomach, cirrhosis of the liver and alcoholic gastritis, of the latter.

Local disease, such as decaying teeth, pyorrhea alveolaris, gangrene, accumulation of secretion in the crypts of diseased tonsils, and cancer of the tongue, may cause persistent foul taste.

Pain is noted in greater or less degree in most inflammatory and ulcerative processes affecting the mucous membranes. It is aggravated by motion of parts, and by contact with particles of food, and especially by the manipulation necessary for a proper examination.

Dryness is noted in mouth-breathers, in diabetes, in fevers, and in nervous individuals under conditions of excitement, especially when attempting to speak in public. Under the name xerostoma a condition is described in which the buccal secretions are so diseased that the mucous membranes become shiny and dry. It is of neurotic origin, and is seen chiefly in women.

The mouth should be examined in a proper light, reflected from a head mirror if necessary, and with a suitable instrument for depressing the tongue or holding back the corners of the mouth. One should note the color of the mucous membrane, especially as regards cyanosis and the pallor of anemia, any coating upon the tongue, local ulcers, syphilitic lesions, growths, diseased or misplaced teeth, enlarged tonsils, elongated uvula, swelling indicating tonsillar or re-



tropharyngeal abscess, herpetic eruptions, and those of small-pox, chicken-pox, measles, scarlet fever and erysipelas. The yellow color of jaundice, and the dark discoloration of Addison's disease, often showing as small round spots, may be noted. Narrowing of the dental arch is common in adenoid disease and in mentally defective children.

The ulcerations may be those of any stage of syphilis, of tuberculosis, or of the various types of stomatitis or malignant disease. False membranes observed may be due to the diphtheria bacillus, pneumococcus, streptococcus, or some variety of leptothrix.

### THE MUCOUS MEMBRANE

**Stomatitis.**—The mucous membrane of the mouth is subject to certain inflammations grouped together under the general name of stomatitis, of which the following varieties are described:

**CATARRHAL STOMATITIS.**—Simple or catarrhal stomatitis is the result of a simple inflammation of the mucous membrane, with redness, swelling, and increased secretion. Irritants, the use of certain drugs such as mercury and iodid of potash, and some of the infectious diseases, may give rise to this variety of stomatitis.

**APHTHOUS STOMATITIS: CANKER.**—Small spots of acute inflammation and swelling are followed by ulceration, with great tenderness and pain upon movement of the parts. The dependence of these lesions upon gastric hyperacidity in certain cases is not as generally recognized as it should be.

**ULCERATIVE STOMATITIS.**—This term is applied to a much severer type occurring in poorly nourished and cachetic individuals. The ulcers are chiefly upon the gums, and cause salivation, fetor and adenitis. Vincent's fusiform bacillus is often present, in association with spirochetes and other organisms.

**MERCURIAL STOMATITIS.**—Mercurial stomatitis accompanies the salivation already described and involves chiefly the gums and the salivary glands. Much soreness, swelling and fetor are noted.

**GANGRENOUS STOMATITIS: NOMA.**—This occurs in debilitated children, usually in asylums. It involves the tissues of the cheek most severely, the affected part turning black, while a red zone of

intense inflammation surrounds it. The gangrenous odor is characteristic. Death from ulceration into an artery is not uncommon. I have observed it in typhoid fever, chicken-pox and measles, and it may occur with other exhausting diseases. It often occurs as a small epidemic in unhygienic institutions.

With gangrenous stomatitis Vincent's spirillum and the fusiform bacillus are often present, and frequently with a mixed infection with the streptococcus, staphylococcus, Klebs-Löffler bacillus, pneumococcus or other organism. Hospital gangrene presents many points of similarity as regards the type of infection.

**PARASITIC STOMATITIS: THRUSH.**—The raised, curdy-looking patches are due to a parasite, the *Oidium albicans*. They may coalesce and cover large patches of the buccal mucous membrane, and are not easily removable. The fungi or the spores are readily seen if the scrapings be mounted in glycerin.

**OTHER FORMS OF STOMATITIS.**—The stomatitis of foot and mouth disease is accompanied by considerable swelling, and the history of a recent epidemic in cattle is usually to be obtained. A stomatitis due to gonorrheal infection is occasionally seen in the new-born, and I have seen it due to the pneumococcus in a foundling baby in the first week of life. Bednar's aphthae appear upon the hard palate in nurslings, and are probably due to the pressure of the tongue or the nipple in the act of nursing. The *perlèche* of French authors is due to the continual licking of the corners of the mouth.

Actinomycosis occurs about a decayed tooth with spread of the inflammation to the gums and to the jaw (big jaw). The yellow ray-fungus is not always readily found. Erysipelas invades the buccal mucous membrane occasionally, having the same characteristics as upon the skin. The eruption of herpes facialis presents much the same appearance as do herpetic vesicles elsewhere. Much pain is noted occasionally.

The ulcer upon the frenum from friction upon the teeth of the enlarged tongue in whooping-cough is quite characteristic. The primary sore of syphilis may be seen anywhere, and mucous patches, described elsewhere, are very common. The ulceration about a gumma is occasionally seen, or the deficiency of tissue left after it. The

effects of the extension of glanders, leprosy, rhinoscleroma and scleroderma to the tissues of the mouth are rarely seen in this country.

**Odor.**—The breath may be offensive because of abscess or gangrene communicating with the air passages, or present in or immediately about the mouth. A foul tongue, pyorrhea, chronic tonsilitis with offensive plugs in the crypts of the tonsils, and various forms of stomatitis are usual causes. The odor of mercurial salivation is fairly characteristic, and of great importance in diagnosis. Many odors due to general disease may be present in the breath without special involvement of the mouth.

**Salivation.**—This is most commonly the result of the toxic action of mercury. If salivation and the odor mentioned under the last heading are present together, the diagnosis of mercurial stomatitis may be made even in the absence of a known source of poisoning. Since mercury is readily absorbed from the skin, rectum, vagina, and bladder, as well as from the digestive tract, the possibilities of poisoning are numerous.

**Hemorrhage.**—Bleeding occurs from the gums in scurvy, in infantile scurvy, and from any part of the mucous membrane in the several forms of purpura. Submucous hemorrhages are seen in many of those diseases in which petechiae occur under the skin. The black tongue of low fevers is so colored on account of capillary bleeding.

**Saliva.**—The saliva and micro-organisms found in the mouth are described elsewhere.

### THE GUMS

The mucous membrane of the gums may be pale in anemia, bluish in cyanosis, and reddened in inflammation. In tuberculosis, cancer, and other cachectic diseases a red line at the margin is noted, but is of no diagnostic significance. In scurvy and infantile scurvy the gums are swollen and bleed readily, and they may ulcerate. In pyorrhea alveolaris pus may be seen escaping from under the detached margin, but swelling of the gums is not a marked feature. When tartar is permitted to collect on the teeth a chronic infection soon causes the gums to retract, and they are more or less inflamed at the free margin.

The lead-line is distinguished by its bluish-black color, and usual limitation to the margin of the gums. Cabot points out that separate dots may be seen rather than a line, and that they occur just back of the free margin. The color is due to lead sulphid deposited in the papillae, and is not present if the teeth be missing. A deposit of carbon is occasionally seen in miners.

Alveolar abscesses and the tumors called epulis are frequently noted. The latter are often sarcomatous, and the jaw may be involved more extensively than the gums.

### THE TEETH

The teeth become loosened after scurvy, pyorrhea, and mercurial salivation, and may thus be lost early in life. If they do not appear in the infant by the end of the first year, rickets or some constitutional enfeeblement should be suspected.

Badly decayed teeth have an important bearing upon the general health because of the consequent inefficiency of mastication, the pain caused as the decay progresses, and the mouth infection resulting.

The transverse furrows upon the teeth indicate that in the developmental stage some cause of general depression of the bodily vigor existed. The eruptive diseases most frequently cause this phenomenon, but stomatitis, mercurial or otherwise, may be the source. Gout causes loss of the polish of the enamel and erosion of the body of the teeth with eventual loosening. The incisor teeth are often badly eroded by acid eructations in peptic ulcer.

**Hutchinson's Teeth.**—In hereditary syphilis the permanent upper central incisors are notched, and narrower at their cutting edge than at the gum. Their color is more yellowish than normal. Although similar deformity can perhaps occur in rickets, the presence of these notches with many of the recognized signs of congenital syphilis confirms the diagnosis, and they should cause us to regard any case as extremely suspicious.

The teeth become stained by the use of iron-containing remedies, but the rapid destruction of the teeth after exhausting illness is due rather to the general bodily depression than to the drugs used.

In fevers accompanied by stupor and mouth-breathing, and most characteristically in typhoid, a collection of filth called *sordes* accumulates upon the teeth. It consists of food remnants, blood and epithelial cells, and bacteria.

### TONGUE

This organ should first be studied as to its size, shape, and general appearance. The anomalous aglossia and microglossia are of little interest, and the same may be said of congenital malformations. Macroglossia depends upon vascular or muscular derangements, syphilis, cretinism, myxedema, acromegaly, the development of a tumor within the organ, or more commonly upon acute glossitis from various causes, or acute edema, at times of the angioneurotic type.

The frenum is occasionally too short. If too long suffocation may result from tongue-swallowing. The swelling and deformity from ranula are of surgical interest, as are carcinomatous and other tumors.

Hemiatrophy of the tongue is seen in facial hemiatrophy, in bulbar paralysis, and occasionally as an associated lesion in chronic anterior poliomyelitis, the lesion in this case being in the hypoglossal nucleus. Taste remains intact. The scars of serious bites of the tongue are of much value in the diagnosis of epilepsy, especially if one cannot obtain a history. I have seen the organ nearly half severed in such cases. A puckered scar with atrophy may follow a gunma.

**Mobility.**—If one hypoglossal nerve be diseased we may have paralysis of the tongue upon that side. In bulbar paralysis both sides are paralyzed, with resulting difficulty in mastication, speech and swallowing, these functions being further impaired by the associated paralysis of the labial, pharyngeal and laryngeal muscles. In diphtheria and paresis the tongue muscles are often affected, and in hemiplegia the tongue may be involved with the facial muscles.

Spasm of the tongue occurs in general convulsions, as in epilepsy, and in tetanus and tetany. Stuttering is in part dependent upon spasm of the muscles of the tongue. In hysteria and disseminated sclerosis it is an occasional symptom.

Tremor of the tongue is noted in typhoid and other asthenic fevers, and in alcoholism. In paresis and bulbar paralysis it is common. Fibrillary contractions may be seen as in other muscular parts involved in the latter diseases.

The slow protrusion and withdrawal of the tongue in typhoid and the jerky movements in chorea are well recognized.

**Discolorations.**—Yellowish patches of xanthelasma are rarely seen, and should suggest the possibility of disease of the gall-passages, as when seen elsewhere. In jaundice the tongue may be yellow.

Staining by fruits, tobacco, medicines, and especially poisons are frequently seen. The black spots of Addison's disease are extremely suggestive of the diagnosis. Ecchymoses, infarcts, telangiectases, and varicose veins are occasionally noted. The pale tongue of anemia and the cyanotic one of advanced respiratory and circulatory diseases should be mentioned. The large red papillae in scarlet fever characterize the "strawberry tongue." The organ may be red and bleeding in hysteria. Black tongue or hairy tongue is characterized by the black discoloration near the elongated circumvallate papillae. In acute glossitis the surface may be shining red, and in other conditions of acute inflammation, especially in the infectious fevers. In geographical tongue there are more or less circular patches in which the epithelium desquamates, and these patches spread and change in form during the chronic course of the disease. It is most common in the young, and is not of syphilitic origin as is often suspected.

Leukoplakia, or smoker's patch, is the term used to define the whitish, smooth, thickened areas seen upon the tongue, generally in males who smoke and who have had syphilis. They may form the basis for the development of epithelioma.

Fissured tongue may result from a superficial glossitis, and there is often a history of syphilis. It may occur without any pathological significance, especially in old men. It is most often seen, aside from syphilis, in liver diseases, and chronic dysentery. Advanced cases are spoken of as "dissecting glossitis."

**Moisture and Dryness.**—The normal tongue is moist from the buccal secretions. These are insufficient to keep it moist, how-

ever, in fevers, under conditions of violent exertion, or great excitement, when decreased in diabetes, hemorrhage, or severe diarrheal diseases, in poisoning by atropia, and in those diseases in which mouth-breathing is to be seen, whether due to the dull mental condition, as in typhoid, and intoxication, or to local disease, as in children with adenoids.

**Pain.**—Pain in the tongue may be neuralgic, rheumatic, or due to a local inflammatory condition. Glossodynia may occur in hysteria or as a part of a tabetic crisis. Smarting or burning of the tongue is common in gastric hyperacidity.

Riga's disease, a papillomatous growth upon the frenum of the tongue in infants, is rarely seen in this country.

**Ulceration.**—The various types of aphthæ have been mentioned. Simple ulcers develop as a result of chronic superficial inflammation of the tongue. Dyspeptic or catarrhal ulcers are frequent in gastric hyperacidity, and cause much complaint because of their sensitiveness. They are small, superficial ulcerations on the anterior portion of the tongue, and are often red and angry in appearance.

Traumatic ulcers result from injury by the teeth or otherwise, and are of interest chiefly because of their importance in the differential diagnosis between ulcer, syphilis, cancer, and tuberculosis.

**SYPHILIS.**—Syphilis shows itself upon the tongue, first, as the initial lesion, generally solitary and upon the anterior portion, and presenting an indurated base. It may be only a fissure, but the induration and the constant glandular involvement are distinctive. Secondly, as mucous patches, generally multiple, and accompanied by others upon the lips and gums. They are grayish white, superficial, and less sensitive than most ulcerations. Thirdly, as a gumma, presenting as a tumor, or as an ulceration of the tumor mass, or later as a scar or sclerosis remaining after healing. Other signs of lues are generally present and confirm the diagnosis of the lingual lesion. The smooth atrophy of the tongue, while generally of specific origin, cannot be regarded as absolutely indicative of syphilis. The red "cobble-stone" tongue results from the fissuring of syphilis.

The pronounced adenitis distinguishes the initial lesion from secondary and tertiary ulcerations in case of doubt.

**TUBERCULOSIS.**—Considering the frequency of tuberculosis, tuberculomata and tuberculous ulcers upon the tongue are comparatively rare. The ulcers may be single or multiple, pale, or with little redness or sign of inflammation, and are usually covered by a grayish viscid secretion. I have known them to exist for years. Almost invariably other manifestations of tuberculosis are to be found, and generally in the respiratory tract.

**CANCER.**—The lesion is single, generally in males of middle or advancing years, painful, chronic, accompanied by slowly developing adenitis, and of great malignancy.

**Coated Tongue.**—The epithelia of the tongue proliferate rapidly, and may accumulate if the organ be subjected to less than the usual traumatism of mastication. One side may be less used because of neuralgia or diseased teeth, and thus show more of a coating than the other. Many healthy people never show a clean tongue, a slight whitish fur being constant, exaggerated if gastric disturbance or other slight indisposition occur.

The pasty coat is seen in those having indigestion, especially if alcoholic liquors be used with an over-abundant diet. It is with this type that the bad taste on arising is commonly found. There is justification for the popular belief that such a tongue indicates an inactive liver, for the effect of a mercurial cathartic is very striking.

In the fevers, and most characteristically in typhoid, we see the dirty, grayish, heavy, moist coating, becoming dry and brown in the height of the fever, or if mouth-breathing occurs. If capillary hemorrhages take place the tongue becomes almost black. The whitish tongue of those fed upon milk, the shaggy tongue of the hypertrophied papillae, and the red tongue of chronic intestinal disease should be mentioned. In general, less attention is paid to slight changes in the appearance of the tongue since the introduction of more accurate and scientific methods for the estimation of the functional capacity of the different components of the digestive system. Yet much general information of great value is readily accessible in the examination of the organ.



### THE LIPS

Double lip, caused by hypertrophy of the labial glands and the consequent eversion of the mucous surfaces, hare-lip, the thick scrofulous upper lip, the thick lower lip of myxedematous idiots, need only to be mentioned. The lips are parted habitually in mouth-breathers.

Tremor is noted in exhausting fevers, in certain neurotic individuals, and in paresis.

Chancre of the lip is more frequent before middle age, and leads to marked swelling and adenitis. Cancer develops as a rule later in life, more slowly, usually upon the lower lip, and is likewise accompanied by adenitis. Angiomata are not common. Fissures are often seen and may become the seat of malignant disease. Herpes labialis is found, especially in colds, pneumonia, malaria, and cerebrospinal meningitis.

The color of the lips varies much from the normal healthy red. We have the pallor of anemia, the ashen pallor of pernicious anemia, the yellow of jaundice, valuable especially in the colored race, the reddening of diabetes in certain cases, the bluish shade of argyria, the cyanosis of cardiac and pulmonary diseases, and of poisoning by coal-tar derivatives, and the intense concord-grape color of chronic cyanotic polycythemia. In a recent case of enterogenous cyanosis, the color was much less deep than in the last-mentioned disease.

The acute swelling of the lip in angioneurotic edema is easily recognized. It is generally attributed by the patient to the bite of some insect, and a similar condition may really occur from such a cause.

### THE FAUCES AND THE PHARYNX

The structures composing the fauces deserve careful consideration because of the frequency of invasion of the body by serious disease originating in this locality. Diphtheria, streptococcic septicemia, infectious arthritis, tuberculosis, rheumatism, purpura rheumatica, chorea, erysipelas and several of the contagious diseases of

children are known or suspected to originate, always or frequently, from invasion of the tonsils or neighboring parts by various micro-organisms.

The passage may be obstructed by enlarged tonsils or by adenoid growths, interfering with respiration and deglutition. Retropharyngeal abscess and disease of the cervical vertebrae may cause obstruction from the posterior aspect. The abscess may result from the vertebral disease. Erysipelas, and sarcoma, or other varieties of tumor may also cause obstruction. The contraction following certain cases of syphilitic ulceration and rhinoscleroma should be mentioned. The passageway is abnormally large in cleft-palate, and in those cases in which the soft palate is destroyed more or less completely by syphilis.

### THE PHARYNX

In acute pharyngitis the mucous membrane is inflamed, at times covered with a mucous secretion, and at others reddened, but with little visible secretion. The latter is typical of gouty and rheumatic pharyngitis. Chronic inflammation is very common and is due to digestive troubles, to the irritation of smoking, and of strong alcoholic drink, and to the improper use of the voice in singing and speaking (clergyman's sore-throat). Inflammation of the organs and cavities related to the pharynx may extend to its membrane. Phlegmonous pharyngitis may supervene upon the acute form. The false membranes of diphtheria and streptococcic inflammation of the throat may extend to the walls of the pharynx. Many of the ulcerations noted as occurring upon the tongue and mucous membrane of the mouth are also found here.

The muscles of the pharynx are not infrequently involved in a rheumatic pharyngitis, difficulty of swallowing being the chief symptom. Paralysis occurs in bulbar paralysis, Landry's paralysis, and other diseases in which the glossopharyngeal and vagus nerves or nuclei are implicated, but is of comparatively little consequence either pathologically or from a diagnostic standpoint, since other much more important structures are involved, so that the pharyngeal paralysis is overshadowed.

Anesthesia of the pharynx is often noted in hysteria, and in the organic diseases of the glossopharyngeal and vagus nerves ((bulbar paralysis, diphtheritic paralysis). Anesthesia with consequent absence of the pharyngeal reflex is a valuable sign of hysteria. Globus hystericus depends upon a disturbance in function of the glossopharyngeal nerve.

Spasm of the muscles of the pharynx is seen most typically in hydrophobia and tetanus. It may be purely hysterical.

### THE PALATE

The palate is subject to many deformities which need not be mentioned here. The most important variation in form is the high, narrow, arched palate, seen in degenerate children.

Attention should be called to the fact that inability to nurse should lead to examination of a new-born baby's throat for cleft-palate. Bednar's aphthae have been mentioned. Diphtheria extends over the uvula and soft palate, and the ulcerations of tertiary syphilis are common, often leading to perforation. Edema of the palate and uvula may become a serious impediment to respiration. Diphtheritic neuritis is the common cause of paralysis of the soft palate, with nasal voice and regurgitation of liquids through the nose as the most prominent symptoms. Herpes zoster occurs here rarely, but is very distressing.

### THE TONSILS

These organs are recognized more and more as the original seat of various serious infections. Diphtheria is generally primary here. More frequent even than this infection with the Klebs-Löffler bacillus is that by the streptococcus, which presents a less typical false membrane, but cannot be positively differentiated excepting by bacteriological examination in certain cases. The false membrane complicating scarlet fever, small-pox, typhoid and measles is generally of this nature. General infection is not rare, and is often of great gravity. I have reported an epidemic of pyemia and multiple abscesses, in which the source of infection in most cases was the streptococcic throat.

Primary and secondary syphilitic lesions are not uncommon upon the tonsils, but tertiary troubles are rare. Retention of secretion in the crypts, associated with an infection with *leptothrix* in many cases, gives rise to the bad-smelling plugs which cause certain cases of foul breath. Tuberculosis and cancer are occasionally seen. *Calculi* may form in the tonsillar crypts.

Acute tonsillitis is one of the most frequent of diseases, especially in children, and gives rise to marked swelling, pain, and fever. The extension of the inflammation to the deeper tissues of the tonsil causes quinsy, or suppurative tonsillitis, generally unilateral, and often the cause of such swelling as to nearly close the pharynx. Chronic enlargement of the tonsils is exceedingly common in children, often as a sequence of repeated attacks of acute tonsillitis. With the associated adenoid vegetations the enlarged tonsils are the common cause of mouth-breathing, with the attendant ear troubles, these originating largely from mechanical interference with the function of the Eustachian tubes. The changes of the voice in quinsy and in chronic inflammation of the tonsils with adenoid vegetations are almost pathognomonic, and should receive the careful attention of the student.

The adenoid growth mentioned causes such obstruction in the posterior nares as to prevent nasal respiration entirely in many cases. The diagnosis may be made by the introduction of the finger up behind the soft palate, but the procedure is a disagreeable one for the patient. The inference is so strong from the facial appearance, voice and respiration that these vegetations exist, that most cases are sent to the specialists without actual physical proof of their existence. The damage to the general health, to hearing, and particularly the increased danger from diphtheria and other infectious throat diseases from the existence of the extensively diseased mucous surfaces found in the cases just described, should lead to their prompt and efficient treatment.

#### ANGINA LUDOVICI

Ludwig's angina is a streptococcic cellulitis, usually secondary to scarlet fever or diphtheria, but occasionally arising from some infec-

tion of the submaxillary gland or the tissues about the roots of the teeth. It affects the region of the floor of the mouth and presents great swelling, induration, and redness of the submaxillary region and the front of the neck, generally ending in suppuration. It is extremely grave. Purely localized abscesses due to the staphylococcus are much less grave in character.

### DISEASES OF THE SALIVARY GLANDS

The ordinary acute infection of the parotid glands, rarely suppurative, is spoken of elsewhere. (*See Mumps.*) As a secondary infection we see parotitis, on one or both sides, most commonly in typhoid, but also in pneumonia, in the various septic diseases and in peptic ulcer. The infection doubtless reaches the gland in some cases through the salivary duct, but probably more frequently by way of the circulation. Suppuration occurs in a majority of cases. The prognosis in any infectious disease is rendered decidedly grave by the occurrence of this complication. In typhoid, however, I have seen recovery repeatedly, the inflammation subsiding spontaneously or the abscess breaking externally (into the external auditory canal in one case), or being opened by the surgeon. In diseases of the generative organs and various abdominal diseases, parotitis may occur, often with suppuration. The salivary glands are all inflamed in mercurial salivation. Iodin preparations, pilocarpin and various drugs may increase the secretion of saliva. Dribbling may occur because of such increase, or simply, as in bulbar paralysis, hemiplegia, etc., because of loss of control over the muscles of the mouth. Ptyalism is an occasional symptom in pregnancy, in disease of the pancreas, and in various infectious fevers.

A striking and unusual disease is the gaseous tumor of Steno's duct or the gland itself, the tumor containing air only, as in glass-blowers, or air, saliva and pus in the case of some infection. The patient is able to squeeze the air out through the natural opening in many cases, causing the tumor to disappear.

A chronic inflammation remains at times after the parotid swelling of mumps and mercurial poisoning, and in syphilis. Chronic

inflammation of the salivary and lachrymal glands and occasionally of the axillary and inguinal glands is found in von Mikulicz's disease. The blood in these cases may show the changes of leukemia or pseudoleukemia.

Inflammation about the outlet of the duct of any one of the salivary glands may be due to the presence of salivary calculus. Cystic, tuberculous, fibromatous, and various malignant tumors of the glands are described in works upon surgery.

### THE ESOPHAGUS

The food is carried from the lower border of the pharynx to the stomach by a muscular tube about  $\frac{3}{4}$  inch in diameter (2 c.m.). Difficulty in swallowing or dysphagia is the chief symptom of disease of this organ, and regurgitation of food occurs if obstruction becomes complete. Normally the narrowest portion of the tube is at the beginning, a point of importance, because of the fact that foreign bodies which pass this location may be expected to pass through the entire digestive canal. Because of its intimate relationship with the trachea, the thyroid gland, the vagus and recurrent laryngeal nerves, the pericardium, aorta, right and left pleura and thoracic duct, injuries and diseases of the esophagus are of especial importance.

We may find nervous spasm, acute esophagitis, rupture, stricture, malignant and other tumors, diverticula, obstruction by foreign bodies, and perforation from within or without in our investigation of the gullet. Paralysis of this organ is scarcely distinguishable from the overshadowing paralysis with which it is associated.

We examine the esophagus by external and internal inspection, palpation, auscultation and percussion, the passage of the sound, and the use of the Röntgen ray.

In the upper left side of the neck we may possibly see and oftener palpate a foreign body lodged in the esophagus, or an abscess resulting from it, or a diverticulum distended with food, or a new growth. Subcutaneous emphysema may signify the perforation of the tube. By the use of the esophagoscope the interior may be

inspected, ulcerations, growths, strictures and foreign bodies located, and the latter occasionally removed. The difficulties of esophagoscopy and the rather infrequent opportunities for its use, tend to limit its practice to specialists.

Auscultation determines practically nothing which cannot be better revealed by other methods of examination. Percussion occasionally serves to locate cancer of the esophagus, when it reaches a great size.

By the passage of the tube, sound or bougie, we may determine the presence of foreign bodies, of spasm of the esophagus, the presence, location, and to some extent the character of stricture, and, by the use of smaller sounds passed successively, the presence of a diverticulum, one or more of the instruments catching in the pocket. The size of the stricture may be determined by using different sized bougies till one is found which will just pass. If an ordinary tube passes readily at one time and sticks fast at another, a diverticulum is probable. Spasm generally yields before a steady pressure. Foreign bodies may be displaced by the instrument used.

Measurements are best taken from the dental arch. The esophagus begins six inches (15 c.m.) from this point, the left bronchus is passed at nine inches (23 c.m.), and the cardiac orifice of the stomach at 16 inches (40 c.m.).

The three places mentioned are somewhat narrower than the rest of the tube.

Alarming spasm of the larynx, even with momentary unconsciousness, may occur upon passing the stomach tube. The terrible respiratory distress arising from its passage into the larynx may not prevent the introduction of liquid food into the lungs in insane patients. One case of puerperal insanity, which I saw, recovered after an intense deglutition pneumonia caused by the introduction of a pint of sterilized milk into the lungs.

Force should be avoided in the use of the sound, and it should never be used without determining the probable absence of aortic aneurism. In one of my cases a fragment of a cancer of the esophagus was withdrawn in the eye of the stomach tube. The presence of blood upon the tube signifies ulceration of some kind,

and should forbid further manipulation. The tube should not be used after recent hemorrhage, and should be used only with great circumspection in arterial disease, pregnancy, acute inflammation of the esophagus, or when varicosities are suspected, as in hepatic cirrhosis.

**Examination by Röntgen Rays.**—Many foreign bodies may be located in the esophagus by the use of the X-ray, and aneurisms or other growths encroaching upon its caliber. By the use of bismuth-containing food, the place of stricture, amount of dilation above it, and form and location of the diverticulum may be determined in favorable cases.

The narrowing found in the caliber of the esophagus may be congenital, spasmodic, or from pressure from without, as by aneurism, or tumor of the tracheobronchial glands, or from cancer, inflammation or cicatricial stricture of the tube itself. Dysphagia from aneurism or large pericardial effusion is not uncommon. Lateral pressure upon the tube by a distended diverticulum must be borne in mind. The local widening of the esophagus from traction of a cicatrix (traction diverticulum) is not capable of clinical diagnosis. Distress under the sternum, regurgitation of swallowed food, cough, absence of the sound of the swallowed food passing into the stomach, emaciation, and the signs found by the use of the stomach tube (*see above*) may determine the presence of a pulsion diverticulum.

**Acute Esophagitis.**—This generally follows the ingestion of irritating or corrosive liquids, of which the lye used in washing, and thus so readily accessible to small children, is much the most common agent. Next to this in frequency in children is perhaps hot water or steam, from the attempt of the child to drink from the spout of the tea-kettle. In adults, corrosive poisons taken by accident or intent are frequent causes. Extension of disease from the throat to the esophagus, as in various infectious diseases, is not uncommon. The acute inflammation around an impacted fish bone or other foreign body often leads to abscess formation around the esophagus, with danger of perforation into the mediastinum, pleura or pericardium.



**Chronic Esophagitis.**—Chronic esophagitis is seen chiefly in those using much strong spirits, and occasionally in heavy smokers.

**Ulcerations.**—Ulcerations of the esophagus are rarely diagnosed clinically excepting when associated with cancer and stricture.

**Cicatricial Stenosis.**—Cicatricial stenosis may follow many of the above conditions, or other pathological processes of which ulceration becomes a part.

Obstruction by cancer constitutes about 90 per cent. of all stenoses. Pain is more frequent in these cases, and, from involvement of the lymph glands, recurrent paralysis with cough and aphonia, and even such tumor formation as to give rise to a marked dullness in the upper sternal region. I have seen cancer of the esophagus and aneurism of the aorta present in the same case with complete obstruction of the gullet.

**Rupture of the Esophagus.**—Rupture of the esophagus may occur spontaneously, as in severe vomiting. Pain, choking, and collapse announce its occurrence. It is very rare. Perforation from too forcible attempts to pass a sound is not very uncommon. I have found by aspiration milk in the right pleural cavity after perforation in the attempt by a careful surgeon to dilate a cicatricial stricture due to swallowing lye.

**Esophagismus.**—Spasm on attempting to pass the tube has been mentioned. The spasm occurring in hysterical and neurotic patients, chiefly of the female sex, and in rabies, may be spontaneous, or may occur upon attempts at deglutition. It may be determined by the steady pressure of a sound, for it yields as does spasm in the urethra. If age, arteriosclerosis or heart disease forbid repeated attempts to pass the sound, we may erroneously conclude that cancer exists. One patient seventy years of age, whom I saw many years ago, starved to death under the diagnosis of cancer, the surgeon properly, according to our best light at that time, declining to force the sound. At the post mortem the esophagus was entirely normal.

**Dilatation.**—Dilatation of the esophagus occurs secondarily above the location of a stricture. Hypertrophy of the muscular walls is present. Dilatation may occur from atony of the walls without the presence of stricture, the sound readily entering the stomach.

## SYMPTOMS PERTAINING TO THE TAKING OF FOOD AND DRINK, TO DIGESTION AND DEFECATION

### APPETITE

The normal appetite varies enormously according to custom, individual taste, season, and especially according to the varying demands made upon the system by different occupations, stress of severe exposure, etc. The normal appetite of a school-girl would not suffice for a lumberman exposed to cold and doing severe muscular work.

**Bulimia.**—Bulimia signifies an inordinate appetite. It is most commonly seen in convalescence from typhoid fever. Diabetics, because of the constant waste of sugar through the urine, often eat enormously. In convalescents from acute mania a prodigious appetite is sometimes present, and bulimia is occasionally seen in other forms of insanity and in hysteria. The hyperacidity of gastric and more especially of duodenal ulcer may cause the patient to eat more freely and more often than in health. The child with whooping-cough eats more than normally in many cases simply because he vomits, in the paroxysms of cough, most of the food ingested.

**Acoria.**—Acoria signifies the loss of the normal feeling of satiety upon eating. It is found chiefly in insane and neurotic patients.

**Anorexia.**—Anorexia signifies loss of appetite so that the thought of food is generally disgusting. It is seen in fevers, in malignant disease, in severe nervous depression, grief, worry and anxiety, in hysteria (anorexia nervosa), melancholia and alcoholism, especially at the beginning of an attack of delirium tremens. In gastric cancer and cirrhosis of the liver appetite is often completely lost.

**Pica.**—Pica signifies a perverted appetite. The perversion seen in pregnant women rarely reaches a degree justifying the use of this term, but in chlorosis, hysteria and insanity, patients sometimes desire chalk, earth or disgusting articles not fit for food. The eating of chalk by young women is probably often prompted by gastric hyperacidity, and the habit is therefore not in these cases to be regarded as disease.

### THIRST

In low fevers, especially in typhoid, because of the mental dullness, thirst is often absent. In most fevers it is increased. In conditions in which much fluid has been lost from the body, as in watery diarrhea, and diabetes, in profuse sweating and after hemorrhage, thirst is a prominent symptom. Acute irritations of the throat, esophagus and stomach often cause a desire for much water. The dry mouth of excitement and xerostomia acts in a similar manner.

### NAUSEA AND VOMITING

Nausea with salivation usually precedes vomiting, but may occur alone. Its causes are, in general terms, the same as those of vomiting, but the associated muscular action of the stomach and diaphragm requisite for emesis is not present.

Vomiting depends upon the excitation of the nerve mechanism in the medulla, the vomiting center, through which a deep inspiration is provoked, the glottis is closed, the diaphragm and abdominal muscles contract, the cardiac orifice of the stomach is relaxed, and the contents of the stomach are violently expelled through the mouth and often in part through the nose. If the stomach be empty, retching results in place of vomiting, and is even more distressing. The contents expelled from the stomach may be mucus, food, blood or the contents of the bowel which have regurgitated into the stomach.

The first matter thus regurgitated after the emptying of the stomach consists of bile and any partially digested food in the duodenum, this accounting for the bitter taste and yellowish green color of the vomitus after the first few attempts at emesis. If the bowel be obstructed, fecal or stercoraceous vomiting occurs, the character of the vomitus depending upon the position of the obstruction. In patients unconscious from any cause, but especially in drunkenness and anesthesia, the inability to close the glottis permits a portion of the ejected matter to be aspirated into the trachea and bronchi, causing suffocation, or aspiration or the so-called deglutition pneumonia.

The vomiting center lies in the medulla close to the respiratory center, and vomiting is produced chiefly through the muscles of respiration. The center may be stimulated to action directly, as by poisons circulating in the blood, apomorphia being the best example; or indirectly by reflex irritation from various peripheral sources. Vomiting is more easily provoked in children because of the great susceptibility of the nervous system and the upright position and underdevelopment of the fundus of the stomach. The regurgitation of an excess of food in babies has little in common with vomiting, the act being free from nausea and other disagreeable sensations.

**Centric Causes.**—In addition to the poisoning of the center by apomorphia already mentioned, centric vomiting occurs from the action of alcohol, ether, and chloroform. The toxic substances circulating in the blood in uremia cause the centric vomiting which is most commonly met with by the clinician. The toxemia of the acute infectious diseases, especially in children, is accompanied by vomiting. While often seen at the onset of pneumonia, diphtheria, small-pox, yellow fever, erysipelas, measles, and mumps, centric vomiting becomes of more diagnostic significance in scarlet fever, for here it is scarcely ever absent. In Addison's disease, diabetes, and various chronic diseases accompanied by toxemia, vomiting may be present.

Vomiting may occur from irritation of the center of origin, or of the peripheral distribution of the pneumogastric nerve. Direct irritation of the terminal fibers in the gastric wall is the most frequent cause, this irritation depending upon the structural damage to the stomach, as in ulcer, cancer, and gastritis; upon pressure from without, as in ascites; upon irritation by emetic drugs, or by stomach contents which are abnormal in character. The impulse to vomit is carried from the center in the medulla to the various visceral muscles concerned, by the phrenic and vagus nerves, and to the muscles of the abdominal wall by the spinal nerves. Pressure upon the vagi by enlarged bronchial glands, as in tuberculosis, and after pertussis, causes a severe cough which is often accompanied or followed by vomiting. Cerebral vomiting depends upon irritation of

the vagus at its origin. This irritation may proceed from meningitis, as in the tuberculous and epidemic forms; from anemia or hyperemia; from concussion; from the pressure of tumor or abscess; from sudden changes in equilibrium, as in car-sickness and seasickness; from Ménière's disease; and rarely as a premonitory symptom of apoplexy. Nausea may be entirely absent in cerebral vomiting. The emesis is sudden and projectile in character, giving the patient little or no relief.

**Reflex Vomiting.**—This may be purely psychical, from a subjective emotion, or a disgusting sight or odor.

**Nervous Vomiting.**—This is seen in neurotic and especially hysterical individuals in whom the stomach and probably the whole nervous mechanism concerned in vomiting are hyperesthetic. The patients may vomit almost everything taken, for weeks together, and still not emaciate markedly. Deception may be practiced and should be guarded against. Juvenile vomiting in neurotic children under nervous strain, and the periodic or cyclic vomiting described by Leyden may be of nervous origin. The latter is probably toxic, may last for a week or more, is often accompanied by hyperacidity and frequently by headache, pain in the abdomen, and constipation. The appendix should be carefully investigated before deciding upon the neurotic character of the vomiting. Those rare cases, one of which occurred in the husband of a patient of mine, in which the father vomits daily during the early months of his wife's pregnancy, are purely neurotic in character.

The nervous type of vomiting so familiar in sick headache should be mentioned. The gastric crises of tabes and other nervous diseases present persistent vomiting, generally with abdominal pain, the latter often very severe. Sharp hyperacidity may characterize the vomitus and it may become blood stained. Such cases have been mistakenly operated upon for gall-stone disease or gastric ulcer, because of the severity of the pain and vomiting.

**Vomiting of Pregnancy.**—In pregnancy and in some cases of dysmenorrhea, uterine displacement, and pelvic tumor, a special type of vomiting occurs. Upon assuming the erect posture in the morning a little mucus is ejected with much nausea and distress. The

vomiting most commonly occurs during the time from the eighth to the twentieth week of the pregnancy, but with very wide variations. I have seen it in the first week and even throughout the entire pregnancy. The patients commonly gain in weight, but pernicious cases occur, leading to a fatal result.

**Vomiting from Faucial Irritation, Etc.**—Vomiting may take place from irritation of the fauces and base of the tongue, especially in hyperesthetic individuals. Foreign bodies, mucus which cannot be readily dislodged, the use of the stomach tube, or the laryngeal mirror may cause it. The paroxysms of severe coughing, as in pertussis and in some forms of tuberculosis, are often followed by emesis arising from faucial irritation. Vomiting may originate in disease or irritation of various abdominal organs aside from the stomach, which we have already considered. In peritonitis, appendicitis, various perforative lesions, hepatic, and renal colic, the different diarrheal diseases, catarrhal jaundice, acute yellow atrophy of the liver, renal and bladder diseases, and in every form of intestinal obstruction, vomiting is a frequent symptom. The sterco-raceous vomiting of obstruction, dependent upon retroperistalsis, is a symptom of the utmost importance in diagnosis.

Vomiting from heart disease is comparatively rare, and may be accompanied by hiccough.

### REGURGITATION

Food, generally the liquid portions only, may be expelled from the stomach owing to the relaxation of the cardiac orifice in conjunction with those muscular contractions which have been described under vomiting. Nausea and distress are absent, and the process is much less violent than vomiting. It is physiological in babies. Food regurgitated from a dilatation or diverticulum of the esophagus does not have the acid reaction normal to the stomach contents.

**Merycism.**—In those patients in whom regurgitation frequently occurs, the habit of regurgitating solid food, chewing it again, and swallowing it is sometimes established, a process normal in ruminat-

ing animals. Rumination is occasionally taught to children by those having the habit.

**Heartburn: Pyrosis.**—This symptom is dependent upon the irritation of the esophagus by the acid contents of the stomach, present by eructation or actual regurgitation. It occurs especially in hyperchlorhydria, but also in neurotic and hyperesthetic individuals when the stomach contents are normal. Its occurrence in hypochlorhydria, not so well recognized as it should be, is dependent upon the presence in the gastric contents of acids of fermentation, these developing because of the lack of sufficient hydrochloric acid to carry on the normal digestion. The regurgitation of sharply acid fluid is the "water brash" of the laity.

**Eructations.**—Swallowed air or the gases of fermentation are frequently belched from the stomach, the gases being frequently offensive in taste and odor. The habit of swallowing air, analogous to "cribbing" in horses, is not unusual, and the eructations in these cases are inoffensive. Belching is very common in various types of indigestion, in hysteria, neurasthenia, certain cardiac diseases, and as a purely vicious habit. It constitutes a polite acknowledgment of the excellence of the host's table among certain races.

### HICCOUGH

The sudden contraction of the diaphragm with passage of air through the glottis, and the characteristic sound, constitutes singultus. Most cases are trivial, but many grave diseases are hastened toward fatal end by persistent hiccough. It is met with most frequently as a grave complication in those diseases of the abdominal and respiratory organs which cause irritation of the diaphragmatic region in some manner, direct or otherwise. Hysteria, gross disease of the brain, and gout, nephritis, and diabetes may also present this symptom.

### DEFECATION

The feces are normally passed daily, but with wide individual variations as to frequency. The average weight is some 5 or 6

ounces, also subject to great variation. The excrement consists of food remnants, enormous numbers of bacteria, mucus, and epithelial cells, and is colored in different shades of brown chiefly by hydrobilirubin. The changes in color due to variations in diet will receive attention later.

The liquid contents of the upper bowel are normally dehydrated in the colon, so that the feces assume a normal consistence by the time of arrival at the sigmoid flexure. If retained too long after this the consistence becomes more firm. The hard, round or oval lumps in the passage indicate that the contents of the pouches in the wall of the bowel have become desiccated beyond the normal point. In some constipated individuals lime salts are even deposited in these lumps. Because of the stercoral ulcers sometimes resulting and the possibility of diverticulitis, these are of importance.

The passage of the feces into the rectum gives rise normally to the desire to defecate. If this stimulus be habitually disregarded, the rectum loses its proper sensitiveness, and the contents remain in it indefinitely, completely abolishing its proper function, for the rectum is designed as a passageway, and not as a storage reservoir.

The internal sphincter is made up of involuntary muscle fibres, and is not under the control of the will as is the external sphincter, yet excessive stimulation may cause the latter to relax in spite of the will. The act of defecation involves the involuntary relaxation of the internal sphincter, the voluntary relaxation of the external sphincter and the contraction of the rectal muscular fibres and of the muscles of the diaphragm with the purpose of increasing the intra-abdominal pressure. This pressure is more efficiently applied in a squatting posture than when seated.

We shall consider deviations from the normal in defecation as follows:

**Constipation.**—This term is used to define abnormal infrequency of defecation, with difficulty, because of the hardness, dryness, and small bulk of the feces. The utmost latitude must be allowed as to the frequency of normal bowel movement, the limits being two or three passages in a day to an interval of one, two, or several days. The large majority of healthy persons have one movement



daily. Persons of dark complexion and bilious temperament are more subject to constipation than others. Constipation may originate from a multitude of causes. Its extreme frequency in civilized life depends largely upon the changes in the manner of living, from that normal to man in an uncivilized or semi-civilized state. The sedentary habits of modern life, the consequent lessened intake of food, the use of foods so prepared as to rid them of their indigestible portions, the introduction of refined sugar containing much nutriment and no residue, the use of tea and astringent wine containing a certain amount of tannin, the frequent lack of desire, because of lack of exercise, for water and other fluid, are some of the causes operating to leave a lessened amount of residue in the bowel and this bland in character, and harder and dryer than normal. Lack of opportunity, in many occupations, for immediate attention to the desire to defecate, is an additional factor.

Constipation may also occur because fever lessens the intake of food and causes the stools to be more thoroughly dessicated; because of lessening in the amount of bile and other digestive juices; because of loss of fluid from the system from sweating or polyuria (diabetes); because of lessened excitability of the motor apparatus of the digestive tract; because of overdistention of the bowel by coarse and indigestible food; because of weakening of the expulsive power of the abdominal muscles from paralysis, overdistention from pregnancy, ascites, etc.; or because of paresis of the intestinal muscles from some inflammation of the peritoneal coat of the bowel.

Local conditions may cause it, as fissure of the anus, hemorrhoids, rectal disease, prostatitis, and disease of the female sexual organs, rendering defecation painful.

Exhaustion of the normal excitability of the bowel may occur from the overuse of purgatives, with resulting constipation. The obstinate constipation of morphin habitués, with fecal impaction in the rectum in some cases, is well known. The tannin in the acorns used by the Digger Indians produces a similar effect. Starvation, either from lack of food or inability to get it into the bowel because of esophageal or gastric disease, may be the cause. Lead poisoning, by setting up a condition of spasm of the intestinal musculature, and

hysteria and pelvic disease in women, accompanied by such spasm, should be noted. In meningitis and various mental diseases, constipation is often present, there being some interference with the nervous mechanism of defecation. Anemic, chlorotic, neurasthenic, and insane patients generally suffer from costiveness. Purely mechanical causes, such as partial obstruction from adhesions, stricture, pressure on the rectum by retroverted uterus, or by tumors, enteroptosis, ruptured perineum, congenital dilatation of the colon or the much rarer congenital dilatation of the small bowel, of which I have reported a case with autopsy, deserve mention. Recent investigations have proved the lessened number of bacteria in the constipated stool as compared with the normal, due to more thorough digestion and absorption of the pabulum upon which they feed. The lessened irritation of the intestinal walls because of their absence is often a factor. The obstinate constipation of certain cases of gastric hyperacidity is presumably due to similar over-thorough digestion, just as the converse state of diarrhea in an acidity may be due to an opposite condition.

Actual intestinal obstruction will be considered elsewhere. The constipated individual suffers from uneasy feelings in the abdomen, and more often from furred tongue, headache, lassitude, and loss of appetite. To the absorption of toxins which should be eliminated, many authorities, following Sir Andrew Clark, attribute the development of chlorosis, and there can be no doubt of the importance of constipation in the development of certain types of anemia and nervous exhaustion. Hemorrhoids, prolapse of the rectum, hypersecretion of intestinal mucus from irritation, neuralgic pain in the rectal region, and even a mild type of jaundice from a gastroduodenal catarrh may result. Diarrhea may occur as the result of fermentation setting in upon the long-retained intestinal contents. The palpation of masses of hardened feces in the colon, sigmoid, and rectum, more especially in women, while making a pelvic examination is familiar to every physician. Dilatation of the bowel, ulceration, perforation, fecal impaction, occasionally with "channelling," and diverticulitis, are grave possibilities in obstinate cases.

The longest continuance of constipation is probably in cases of

congenital dilatation of the colon. In one such case coming under my observation the patient had no stool for three months and then passed nearly 30 pounds of feces when the bowels were finally opened. Many similar cases have been reported.

**Diarrhea.**—In this condition the stools are more frequent and more nearly liquid than normal.

Diarrhea may depend upon increased peristalsis alone, which may be due to purely nervous influences, as in the diarrhea occurring from fright or anxiety, and in hysteria, exophthalmic goiter, and the abdominal crises of tabes. The intestine presents no pathological change, and the stools are simply more fluid than normal.

Increased peristalsis is a prominent factor in the diarrhea arising from the use of purgative drugs, and from various irritants to the intestinal mucosa, whether introduced from without, as improper food, arsenic and other poisons, taken by the mouth, or irritants, given by enema, or generated within the digestive tract, as in the fermentation of certain foods, either because of the nature of the food or because the digestive power is affected. This type is especially frequent in children, and is associated with the presence of various micro-organisms and toxins due to their action.

The diarrhea resulting from overeating and drinking should be mentioned. Catarrhal inflammation is a factor in many cases of the type just described.

The bowel wall may be irritated, peristalsis increased, and in some cases a catarrhal inflammation established, because of the presence of some constitutional disease. Thus the eliminating of urinary waste products by the bowel occurs in uremia, with resultant diarrhea. In sepsis, after the extensive irritation of the skin, as in burns, and in various infectious diseases, this symptom is often present. Diarrhea occurs when the amount of fluid within the intestinal canal becomes greater than normal, whether from direct increase, as after the use of saline drugs and in cholera, or from failure of proper absorption, as in the portal congestion of hepatic cirrhosis.

Diarrhea is a symptom of many varieties of intestinal ulceration, as in typhoid, tuberculosis, and dysentery, and the ulceration of

malignant disease. The presence of blood and mucus is commonly noted in many of these cases.

The diarrhea of mucous colitis is accompanied by the passage of mucous casts or shreds, and is especially seen in neurotic women. In the stools of the lenteric diarrhea of the older authors undigested fragments of food are a prominent feature. If the intestinal contents escape prematurely, as through an artificial anus or fecal fistula, or if the canal becomes "short-circuited" by a pathological or surgical communication between the upper and lower portions, diarrhea often results.

The association of the bacillus coli, *B. enteritidis*, *B. dysenteriae*, *Entameba histolytica*, and other organisms, with various types of diarrhea will be discussed elsewhere.

The stools may number from a few to a score or more in the 24 hours. The earlier movements in acute cases contain chiefly food remnants, the later ones mucus, increased amounts of intestinal secretion, and often blood, frequently only in traces. Pain, abdominal distention, flatulence, tenesmus, irritation about the anus, thirst, diminution in the amount of urine, weakness, faintness in some cases, and even collapse with lowered temperature, may be noted. Fever is a frequent accompaniment, and vomiting is often present. If much fluid be lost, as in cholera nostras and Asiatic cholera, cramps in the muscles, especially of the legs, are complained of.

Chronic diarrhea is usually associated with some severe catarrhal or definite ulcerative lesion in the bowel wall. Chronic intestinal catarrh is the basis of the morning diarrhea seen in neurotic individuals, in which several loose passages occur during the forenoon, abnormal only in their soft consistence and in the excess of mucus.

**Tenesmus.**—The painful straining at stool which develops in the course of diarrhea is called tenesmus. This symptom may also occur in dysentery, and in almost any disease producing an irritation of the rectal mucosa. This may be a simple proctitis, or the result of trauma, or from malignant disease, prostatitis, cystitis, especially in association with vesical calculus, or from a polyp, intestinal parasites or impacted feces. Prolapse of the bowel often results

from the severe and ineffectual straining, and hemorrhoids are a frequent sequel.

Other causes of pain in defecation are to be found in the mechanical conditions in the rectum and anus. The passage of a large and hard lump of fecal matter in constipation may start a fissure, which gives pain whenever the sphincter is stretched. Inflamed hemorrhoids, ischiorectal abscess, fistula, enlarged prostate, a retroverted womb, especially if endometritis or pregnancy be present, and syphilitic, malignant, or other ulcerative processes cause pain. Very acute pain is present in the rectal crises of tabes, without the signs of local disease. By causing the patient to defer the emptying of the bowel for fear of pain the conditions are made worse by the accumulation of hardened feces.

**Incontinence of the Feces.**—The contents of the bowel, especially if liquid, escape when the sphincter is inefficient, either because of interference with its nervous control or its mechanical structure. The brain may lose its general control, as in coma, or the center in the cord, as in myelitis, or the sphincter may have been torn in labor, or damaged in rectal operations, or by syphilis or malignant disease. In cholera, typhoid, and other general diseases the trouble may be due in part to the affection of the nervous system, and in part to the utter weakness of the muscles of the sphincter. The relaxation of the sphincter from fright is not uncommon, and is noted also in epileptic convulsions, sunstroke, and other brief periods of unconsciousness.

### GASTRO-INTESTINAL RÖNTGENOLOGY

“Gastro-intestinal röntgenology depends mostly upon a study of serial, instantaneous plates made after the ingestion of food containing an opaque substance. My method is a combination of the Holtz-knecht and Haudek six-hour-interval method and the serial röntgenography of L. Gregory Cole. The fluoroscope is of minor, but not of minus, importance.

“Various lesions of the gastro-intestinal tract lead to abnormalities in position, size, outline, and motility of the digestive organs; these

are indicated upon the plates, and when properly read and correlated may be translated into terms of pathological condition.

"The röntgenologic examination of the digestive tract is much to be preferred to exploratory laparotomy, as the inherent dangers of anesthetic and operation are avoided; some appearances indicative of pathology which are shown upon the plate are not to be found in an anesthetized patient.

"One can readily see that a stomach filled by a bismuth meal, if ptosed or dilated, will cause appearances upon the plate such that the most untrained observer may identify the condition; many other conditions are not so readily discernible; the work demands a skilled and trained röntgenologist."—G. H. STOVER.

## SECTION V

### EXAMINATION OF THE BODY FLUIDS

#### GASTRIC CONTENTS

##### MACROSCOPICAL EXAMINATION

**Stomach Contents.**—The stomach contents obtained with the tube after giving a test-meal should be examined macroscopically as to quantity, consistence, color, odor, presence of blood, mucus, food remnants, etc.

**QUANTITY.**—Forty-five to sixty minutes after the customary Ewald test-meal, we may expect to obtain an ounce or two (30 to 60 c.c.) of stomach contents. In cases of increased motility none may be present, while if the secretion of gastric juice be abnormally large or the pylorus be blocked, five or ten times the normal may be obtained. Even a liter of practically pure gastric juice may be present in the fasting stomach of hypersecretion. If the precaution be not taken to wash out the dilated stomach of pyloric stenosis, before giving the test-meal, as much as five liters may be evacuated with the tube, as in one of my cases. The presence of food particles known to have been taken six or seven hours before indicates delay in the emptying of the stomach, since any meal should have been disposed of in this interval.

**CONSISTENCY.**—This varies from that of water to that of mush. In a general way we assume liquid gastric contents to have a fair or high acidity, while a mushy consistency is found often with achlorhydria. Mucus may be so abundant as to cause the whole contents to move as one mass if the vessel be tipped. If the digestive power of the stomach be low, particles of the toast of the test-meal may appear like water-soaked bread, and bits of the crust may be distinguished, whereas the digestion should have been so complete as to reduce all to a puree-like mass.

**COLOR.**—If the gastric juice be obtained practically unmixed,

it is almost colorless. The usual contents are rather whitish or grayish and opaque. Much mucus gives a white-of-egg appearance. Brownish specks result from the presence of bits of crust of the bread eaten; or of the meat in certain test-meals.

The most significant color is the bright red of fresh blood and next to that the brownish color of the blood altered by the digestive processes. The former is more characteristic of ulcer, the latter of cancer, but with very wide variations. A yellowish or greenish color is often seen from bile, but much more commonly after vomiting than in a test-meal. Pus may be present from perigastric suppuration, being recognized if in any amount by its usual color.

**ODOR.**—A slightly sour odor is normal. If food be long retained decomposition gives rise to a sour or fatty-acid smell, or even a very offensive one, especially in cancer. The odor of alcohol or of medicines taken should not be found in a test-meal.

Particles of food, taken at a previous meal, blood clots, mucus, and rarely fragments of tissue, may be seen in the contents after the test-meal. I have once obtained a cancerous fragment large enough for histological examination.

**Vomit.**—The appearance of the vomitus varies much more widely than that of the contents after a test-meal. The amount may be enormous if the pylorus be occluded. The consistency may be anything between that of water, as in regurgitated contents of acute dilatation of the stomach, or the gastric juice, to that of mucus or the mushy vomitus from the stomach of low digestive power. The color may be any of those of the test-meal described, and is more frequently altered by the presence of bile, especially after the first efforts at emesis. The fecal vomitus of obstruction is usually brownish in color. The rupture of an abscess into the stomach or esophagus gives the color of pus, perhaps bloody, and occasionally of the color of the pus of hepatic abscess. Various foods and medicines may give different colors to the vomitus. The odor varies much more than that of the test-meal. It may be intensely sour, if retained food has fermented; fecal in stercoraceous vomiting, and more offensive according to the distance of the obstruction from the pylorus; ammoniacal in uremia; putrid in case sloughing has occurred



in or adjacent to the stomach; and, finally, the odor may partake of that of food or drugs taken, be it that of onions, alcohol, ether or other anesthetic, carbolic acid, hydrocyanic acid, or other poisons, spiced foods, or other odoriferous substances.

Various constituents of the vomitus attract attention, especially particles of undigested food, curdled milk, blood, pus, mucus, sputum which has been swallowed, especially in children, round worms, segments of tapeworm, fragments of echinococcus cysts, or other parasites and foreign bodies. The finding of particles of food eaten many hours or several days before points to deficient gastric motility, generally from an obstruction at the pylorus. In a general way retention of particles of the evening meal until the test-meal is given next morning signifies a definite "surgical" obstruction.

The reaction is usually acid, but may become alkaline from blood, bile or saliva, or in uremia. The bitter taste of bile and the sour taste of hyperacidity are often mentioned by the subject.

Especial mention should be made of the brownish watery vomitus of peritonitis; of the suddenly developing fecal vomiting due to perforation of a cancer of the stomach into the bowel, with regurgitation of the intestinal contents through the opening; of the black vomitus from partly digested blood in yellow fever; and of diphtheritic membrane which has been swallowed, or is forced out by the strain of vomiting.

#### MICROSCOPICAL EXAMINATION

After the usual test-meal one may find with the microscope, in the stomach contents, starch granules, epithelial cells, possibly a few red and white blood-cells, and bacteria, often introduced with the food. In contents from meat-containing test-meals, meat fibers, fat droplets and vegetable remnants may be present. In vomitus there may be also blood or pus in abundance, shreds of tissue, yeast cells, sarcinæ, crystals of bile acids, fatty acids, calcic oxalate, cholesterin, and occasionally, in alkaline contents, triple-phosphate crystals. Various protozoa, especially flagellate and amebic forms, and monads are found, particularly in the vomitus from non-obstructive carci-

noma. In cholera the comma bacillus is occasionally seen in the vomitus.

The Oppler-Boas bacillus indicates that lactic acid is present, and, since this is a frequent accompaniment of fairly developed gastric cancer, the finding of this organism attains considerable importance. It is found in approximately 80 per cent. of all cases of the disease. If absent in the contents of a dilated stomach, we should suspect a benign stenosis rather than a malignant one.

Sarcinæ indicate stagnation of gastric contents, with fermentation. Yeast cells often appear in the same specimen.

Simon states that the presence of protozoa in the stomach contents is suggestive of non-obstructive carcinoma.

### CHEMICAL EXAMINATION

Since the gastric juice is largely dependent upon its HCl content for its digestive power, the determination of the amount of free acid, combined acid, and acid salts assumes first importance. The normal free acidity is due to HCl, though at times lactic, acetic, butyric and at times other free organic acids are present, especially when low HCl acidity permits of fermentation of the stomach contents. The combined HCl is in chemical union with protein substances.

**Qualitative Tests.—FREE ACID.**—The presence of free acid may be quickly determined by the use of Congo red paper or solution, the intensity and rapidity of the blue reaction roughly indicating the acidity. It should be noted that watery contents bring about a sharper reaction than we obtain if mucus be present or if the food be mushy in consistence, since the paper is more quickly saturated. HCl produces a sharper reaction than the less active organic acids.

Having determined the presence of free acid, we may apply the dimethylamido-azobenzol test to determine if it be HCl, which gives a reddish pink color, deeper in proportion to the fluidity of the contents, the absence of mucus and saliva, and the amount of free HCl. The test is not absolute, however, since other acids, organic or inorganic, may cause the reaction. This reagent should be more com-

monly used in the form of a *test paper*. If freshly prepared, one may form a fairly accurate opinion of the digestive strength of the gastric juice by the character of the reaction. Even a drop of contents in the eye of the stomach tube may suffice to give a fair idea of the digestive activity of the stomach, since the determination of a proper amount of free HCl is the single most important feature in gastric analysis.

**LACTIC ACID.**—Uffleman's test is the one recommended by most authorities. To 20 c.c. of 10 per cent. solution of carbolic acid is added one drop of 10 per cent. solution of ferric chlorid, the blue mixture resulting being diluted with water until it changes to a light amethyst. The appearance of a yellow color upon the addition of a few drops of the stomach contents signifies the presence of lactic acid. If a large amount of the contents to be tested be used the result may be confusing. The student should try out this test with a standard solution of lactic acid before depending too much upon his results. The extraction of the lactic acid by ether gives a much clearer reaction. If a few cubic centimeters of the stomach contents be "layered" under the Uffleman's solution, the resulting colors are much less liable to cause confusion, since the yellow of the contents is usually different from that resulting from the presence of lactic acid. The presence of lactic acid depends upon a bacterial action not consistent with the presence of the normal proportion of HCl.

**VOLATILE FATTY ACIDS.**—If present in any notable quantity, butyric and acetic acids may be detected by the odor. Their frequent presence signifies stagnation and fermentation of gastric contents.

**Quantitative Tests for HCl.**—The methods given by Sahli are probably the most convenient, accurate and generally applicable that we have. The following directions are modified from his more extensive presentation of the subject. The following solutions are necessary:

(1) A 0.5 per cent. alcoholic solution of dimethylamido-azobenzol. This indicator is sensitive only toward free acids, and is therefore used in the determination of the free acidity, generally due to HCl;

(2) A 1 per cent. aqueous solution of alizarin for the determination of the combined acidity;

(3) A 1 per cent. alcoholic solution of phenolphthalein for determining the total acidity, since this indicator reacts to free and combined acid and to acid salts;

(4) A  $\frac{n}{10}$  NaOH solution.

"The total acidity is found by titrating 5 or 10 c.c. of the gastric contents with the  $\frac{n}{10}$  NaOH solution after the addition of 3 drops of phenolphthalein. The titration is continued until the pink color, which becomes permanent near the end of the reaction, can no longer be deepened by the further addition of a drop of the  $\frac{n}{10}$  NaOH solution. The number of cubic centimeters required for the end reaction corresponds to the total acidity."

"The combined acidity is determined by adding three drops of the alizarin solution to 5 to 10 c.c. of the gastric contents and titrating with the NaOH solution. The color of the solution, when the end reaction is reached, is pure violet. The red color which appears after the yellow of the indicator has disappeared must be completely removed, and the color must be a decided violet."

"The difference between the number of cubic centimeters found in this titration and that of the total acidity equals the combined acidity of the gastric contents."

"The free acid is estimated by taking 5 to 10 c.c. of the gastric contents, adding 4 drops of dimethylamido-azobenzol, and titrating with the  $\frac{n}{10}$  NaOH. The titration is continued until the pink has disappeared and the color has become greenish yellow. This number of cubic centimeters is equivalent to the free acidity."

"The value of these various acid factors can be expressed in many ways. If it is required that they be in terms of HCl, then it is simply necessary to multiply the number of cubic centimeters found in the titration by 0.00365. The product will equal the number of grams of HCl in the volume of the gastric contents employed for the titration. If percentage is required, the above product must, of course, be further multiplied by the fraction  $\frac{100}{x}$ , where x equals the cubic centimeters employed in the titration.

As Prof. Sahli has suggested, it is more usual to express these

values in terms of the number of cubic centimeters of the  $\frac{n}{10}$  NaOH which are required to complete the respective end-reactions for 100 c.c. of gastric contents. If 10 c.c. of the stomach contents are used, the number of cubic centimeters used up in titration is multiplied by 10, etc.

"In cases where only small volumes of gastric contents stand at the disposal of the investigator, Einhorn's modification may be found to be very useful. It consists in the performance of the dimethylamido-azobenzol and phenolphthalein reactions upon the same portion of contents.

Ten cubic centimeters are measured off, and to these are added three drops of phenolphthalein and 4 drops of the dimethylamido-azobenzol. Since the former is colorless in acid solution, the color of the mixture is entirely dependent upon the latter indicator, and the titration may be carried on with the  $\frac{n}{10}$  NaOH in the same manner as in the determination of free acid, i. e., to the appearance of the greenish yellow color. The reading is then made upon the buret, and the  $\frac{n}{10}$  NaOH solution further added until the pink color appears and allows of no further deepening upon the addition of another drop of the alkali. This second reading will correspond to the total acidity, and the first reading to the free acidity, when both readings are made from the original position of the fluid in the buret." <sup>1</sup>

**Tests of Digestive Power.**—This function depends upon the amount of pepsin and of free acid, especially free HCl, and is tested solely by a process of artificial digestion. For this, fibrin stained with carmine, which latter is released by the process of digestion, and gives a red color to the solution, may be employed (Grutzner) or disks of coagulated egg albumen. The proteid body is placed in a tube with a few c.c. of gastric juice and kept warm in an incubator. If the digestive power be normal the rounding of the edges of the particles may be detected in one or two hours, though many hours may be needed for complete solution. These tests are but little used in practice, since the digestive power is nearly proportional to the amount of free HCl, and this may be determined more easily than the power of pepsin alone.

<sup>1</sup> Sahli: "Diagnostic Methods."

**Rennin Ferment.**—This possesses the power of coagulating milk independently of the presence of acid. If one part of gastric filtrate be added to 20 or 30 of milk and a body temperature maintained, coagulation should occur in 10 or 15 minutes, junket being formed. Absence of free HCl does not interfere with the test. Rennin zymogen is quite resistant to alkalies, and is converted into rennin by the action of acids.

**Mucus.**—The small amount normally present is easily recognized, being present as small flakes. Mucus from the pharynx may be swallowed and appears in larger masses, often recognized macroscopically. That from the bronchi is distinguished microscopically by the presence of characteristic epithelial cells, and absence of food particles.

In gastric catarrh, we find the amount of mucus increased, and it contains many epithelial and polynuclear cells, or their nuclei only, if they have been much altered by strong gastric juice. If HCl be diminished the mucus appears to be in larger quantities than otherwise, since it is not dissolved as it should be, or even actually swells in the complete absence of HCl. No quantitative test is available and one would be of little value, since the macroscopic estimation suffices so well. This is especially easy in performing the quantitative tests for HCl in which the mucus shows very distinctly.

### RESULTS OF EXAMINATION

In the examination of the test-meal the results of most importance to the clinician are fortunately to be obtained without serious trouble or expenditure of time. They may be stated as follows: (a) *The Quantity.*—The quantity is of especial importance if greatly increased over the normal one or two ounces, since it indicates a deficient power of the stomach to empty itself. The diagnosis of pyloric obstruction may commonly be made solely through the finding of a large amount of retained and fermented food in the stomach. If the stomach be dilated or its motility be thought to be low we may give two or three figs or other seed-containing food the evening before the test-meal. The finding of the seeds the next morning is good evidence of inability of the stomach to properly empty itself. (b) *The Consistency.*—Strongly acid contents are commonly watery, while pasty,

mush-like contents indicate low acidity, and feeble digestion. In this case undigested remnants of food are present. (c) *Presence of Visible Blood*.—This commonly indicates ulcer if fresh, and cancer if partly digested. Chronic gastritis, hemorrhage from veins of the esophagus, swallowed blood, etc., must be thought of. The chemical detection of blood is of less direct diagnostic value. (d) *Presence of Excess of Mucus*.—This is to be taken as indicating a chronic inflammation of the gastric mucosa, and is more commonly found in cases of low acidity, although in acid mucous gastritis it is often very abundant. (e) *Acidity*.—The presence of a fair or excessive amount of HCl acidity may be determined with tolerable accuracy by the use of the dimethylamido-azobenzol test-paper. The paper must be fresh and have been previously tested. The examiner must remember that watery contents give a quicker and sharper reaction in proportion to the acidity than thicker contents, and that the presence of mucus decidedly interferes with the reaction. (f) *Notably Sour Odor*.—This may be taken in general to indicate that fermentation has taken place, this leading to the inference that the normal gastric acidity was not present, since it tends to inhibit fermentation. (g) *Presence of Bile, Etc.*—Presence of bile, as rarely in duodenal stenosis; of pus, shreds of tissue, fecal matter, gall-stones, parasites, alcohol, poisons, etc.

**Motility of the Stomach.**—Defective motility, if marked in degree, will already have been recognized in the physical examination of the abdomen, through the splashing detected, in most cases. The large amount of sour vomitus mentioned under test-meals constitutes proof of it. In the absence of such proof, we may give one gram of salol well mixed with the test-breakfast. Salicyluric acid should be detected in the urine in 75 minutes. If marked insufficiency exists the salol will not be entirely eliminated in the 24 hours which constitutes the normal time limit. The longer after this time the acid can be found, the more marked is the insufficiency.

For the more intricate examination of the stomach contents the reader is referred to the larger works on the subject.

**Pyloric Stenosis.**—The retention of food in large quantities in the stomach indicates defective gastric motility, but not necessarily either

reflex or organic pyloric stenosis. Sahli has used a cork ball one centimeter in diameter in testing for stenosis, since if it can be found in the stool the next day the pylorus must be open. In general the decision as to whether organic stenosis exists rests upon deductions from the history and examination of the patient. If ulcer be present, or there is evidence of its previous presence, or if pyloric cancer seems probable, the delayed emptying of the stomach is probably due to structural change in the pylorus. If gall-stone disease or appendicitis be present or strongly suspected, it is best to give especial consideration to the possibility that delayed emptying of the stomach may be due to pyloric spasm originating in the regions mentioned. Fortunately the surgical exploration called for in most cases of serious and prolonged gastric retention will furnish the exact diagnosis.

**Defects in Gastric Juice.**—The characteristics of the gastric juice in disease should be recapitulated:

**HYPERCHLORHYDRIA.**—By this term we mean an excess of free HCl, generally associated with a high total acidity. Few individuals in health are comfortable if the HCl passes 50 degrees, and for any but those doing severe work and demanding vigorous digestive power, this mark is probably too high. We should recognize a relative hyperacidity. For certain sensitive neurotic individuals 40 per cent. acidity is too high, and the patient is more comfortable with an antacid and a proteid diet.

We should recognize that hyperacidity is only a symptom, and although in neurotic patients it may constitute the whole of the disease, its more frequent association with gastric ulcer must not be overlooked. Many cases depend upon reflex disturbance from chronic appendicitis and gall-stone disease and I have seen many recover from the symptoms upon removing the reflex cause by operation. The stomach may dilate enormously because of the pyloric spasm in association with a long continued hyperacidity.

**ACHYLIA GASTRICA.**—The defect in the gastric juice with absence of HCl, and the digestive ferments, very low total acidity, and utter lack of digestion, as shown by inspection of the contents after a test-meal, may be due to atrophy of the gastric mucosa, as is often



seen in grave chronic gastritis and carcinoma, or to functional disturbance, as in pernicious anemia. Since anemia follows the organic type, however, care must be used in distinguishing between cause and effect. Motility is commonly little impaired and hypermotility may be present.

**GASTROSUCCORRHEA.**—If the gastric juice be secreted in excess of the normal demands of digestion it constitutes such a hypersecretion as is designated by the term gastrosuccorhea. The condition is present if we find an excess of gastric juice in the fasting stomach when no stenosis exists. To make certain of its existence we wash out the stomach at night and determine the amount of gastric juice the next morning by the use of the stomach tube. If it exceeds one or two ounces (30 to 60 c.c.) in quantity, is acid and contains no food remnants nor other evidences of retention, the diagnosis is established. Spasm of the pylorus from irritation may later lead to dilatation of the stomach. The disease is probably a functional neurosis. If intermittent it constitutes the so-called Reichmann's disease.

**NERVOUS DYSPEPSIA.**—The diagnosis of this disease is established upon other evidences than that obtained by examination of the stomach contents. The results of such examinations are so inconstant and show so little that is definite and tangible that the examination is of value in excluding organic disease rather than otherwise.

**ULCER OF THE STOMACH.**—I have used the stomach tube in more than 300 private cases of ulcer without the slightest mishap, and therefore feel that if used with care and judgment, no contra-indication to its use exists, excepting recent hemorrhage. A high acidity is often found, and blood is present in the contents in probably 10 per cent. or 15 per cent. of the cases. I have repeatedly found hyperchlorhydria in ulcer which had already become carcinomatous, and even in cases which had passed the operative stage, and on the other hand, have found the acidity below normal in many cases without finding cancer upon operation. The test-meal takes a rank entirely subsidiary to that of the history and the clinical examination in this disease. Even when pyloric obstruction exists and several

pints of contents are recovered after an Ewald test-breakfast, a high acidity of the entire contents is not inconsistent with extensive cancerous disease upon the base of an obstructing pyloric ulcer, as I have seen proved in the operating room.

**CANCER.**—Absence of free HCl is the most distinctive feature of the chemical examination of the contents in cancer, occurring in 80 per cent. or 90 per cent. of the cases. Cancer so frequently develops upon ulcer that the acidity is often in a way a compromise between the high acidity of ulcer and the anacidity of cancer. The absence of HCl in a case presenting other signs and symptoms of cancer is a valuable point in diagnosis, while its presence, even in normal amount, cannot entirely exclude malignant disease. The presence of blood and lactic acid, and the finding of the Oppler-Boas bacillus, with absence of HCl, renders the diagnosis of cancer extremely probable. Lactic acid is present in even a larger proportion of cases of cancer than we find to have absence of free HCl. Oppler-Boas bacilli are present in over 75 per cent. of the cases of cancer.

The fact that HCl is often absent in tuberculosis, in pernicious anemia, in chronic gastritis, and other diseases, should render us cautious in estimating the value of this finding in any individual case.

**ACUTE GASTRITIS.**—We rarely need the tube in cases of acute gastritis, since the vomitus shows undigested food of considerable acidity, and with much mucus, and occasionally traces of blood. The signs and symptoms are of more value than the laboratory examination here.

**CHRONIC GASTRITIS.**—The contents show lack of digestive power upon the part of the gastric juice, being often thick and pasty, mixed with much mucus, and giving no reaction or a feeble one to the test for HCl and activity of the ferments. The acidity and motility of the stomach vary much in different cases. If steady loss of weight ensues, cancer must be suspected. Chronic gastritis of alcoholic origin is especially characterized by large amounts of stringy mucus, which are often vomited upon rising in the morning. Owing to chronic inflammation of the gastric mucosa, many pus cells and epithelia are to be found with the microscope, especially in the mucus.

**Hour-Glass Stomach.**—This may be strongly suspected when two portions of stomach contents obtained from the same test-meal vary greatly in character, or when after presumably emptying the stomach with the tube, a considerable additional quantity of contents is obtained. The diagnosis is practically settled if this occurs, and the second portion differs from the first.

### THE VOMITUS

There may be ejected in vomiting the food, mucus, pus, or blood which has reached the stomach through the esophagus, or the contents of the bowel or the gall-bladder regurgitated from below, or fluids which have entered the stomach through a perforation from without its cavity. Parasites which have developed in the stomach or entered it from the bowel should also be noted.

The vomitus varies in amount from a mouthful to much more than a gallon. If hyperacidity be present it is sour and burning; if the food has been retained long it is often so changed by fermentation as to be most disgusting in taste and odor. Different foods, medicines and especially poisons may give characteristic tastes and odors. A urinous and ammoniacal odor is common in uremia. Blood has a salty taste. Bile, saliva and blood tend to give an alkaline reaction to the vomitus, which is otherwise generally acid.

The first vomited matter consists of whatever happens to be in the stomach, generally more or less mixed with mucus and saliva secreted as a result of the nausea which precedes the vomiting. If food has been recently taken it is little changed, while if the stomach has failed to empty itself normally and digestion has been arrested, a similar vomitus may be ejected many hours after eating. Mucus is generally abundant in this case. Curdled milk shows that the ferment has acted properly, while the vomiting of milk unchanged often indicates serious gastric disability.

After the stomach is emptied, if retching continues, the gall-bladder is emptied into the duodenum and the bile then enters the stomach, giving rise to the bitter yellowish-green or bilious vomiting. If obstruction of the bowel be present, fecal vomiting follows, becom-

ing more offensive as lower portions of the bowel are successively emptied.

Watery fluid is ejected if the stomach contains no food, generally mixed with mucus. It indicates the presence of water recently ingested, of saliva swallowed, of gastric juice or mucus or combinations of these. If the vomitus contains hydrochloric acid in normal amount and is abundant, there is present hypersecretion; if the acid exceeds the normal 0.2 per cent. there is hyperacidity. These conditions are common in gastric neuroses, gastric ulcer, the gastric crises of tabes, in migraine and other nervous disturbances.

A large amount of mucus is found in chronic gastritis, especially the alcoholic form, and particularly when vomiting occurs in the early morning. The vomiting of large quantities of watery fluid is seen in acute dilatation of the stomach, and should demand immediate investigation, especially after abdominal operations.

### HEMATEMESIS

The vomiting of blood is an important feature in the diagnosis of many different conditions. The blood is either fresh or recently clotted, or partially changed by the digestive fluids (coffee-ground vomitus). It is never pink or frothy, as is the blood in hemoptysis, and is raised by vomiting or regurgitation and not by coughing. Tarry stools may not help us in the diagnosis, even if noted, for it is possible for the blood of pulmonary origin to be swallowed and give rise to them.

The vomitus is often streaked with blood as the result of straining in the act of vomiting.

The blood ejected from the stomach may have been swallowed, as by a nursing baby from a cracked nipple; by an epileptic from his bitten tongue; by a patient with epistaxis, or by a malingerer with purpose to deceive. It may reach the stomach from the vessels of the esophagus, stomach, bowel or neighboring organs. It may enter the stomach from the bowel, in duodenal ulcer, or thrombosis of the superior mesenteric vessels. In diseases accompanied with obstruction of the portal vein, of which cirrhosis of the liver may be taken as a type, the engorgement of the veins in the gastric and lower esopha-

geal mucosa often results in hemorrhage. In visceral engorgement of cardiac disease we may have the same result, and occasionally in leukemia, splenic anemia and other diseases causing great enlargement of the spleen.

Aortic or other aneurisms may rupture into the stomach or esophagus and cause fatal loss of blood.

Traumatism especially by kicks, blows and crushing injuries, may cause even fatal hematemeses. I have seen fatal vomiting of blood from the giving way of improperly prepared catgut after gastro-enterostomy.

After the ingestion of corrosive sublimate, lime, mineral acids or other corrosive poisons, blood may be ejected. The black vomitus of yellow fever consists of partially digested blood, and the thin dark vomitus of peritonitis is from the same cause. Minute erosions often occur in the gastric mucous membrane after severe abdominal operations, and especially when the omentum is wounded, and the dark vomitus may receive its color from hemorrhage from these erosions.

In those diseases accompanied by bleeding under the skin and from the mucous membranes, such as malignant small-pox, hemorrhagic measles, typhus fever, the purpuric group, hemophilia, leukemia, severe anemia, and occasionally in the new-born, hematemeses occurs.

It is an open question whether the alleged hysterical bleeding and vicarious menstruation do not proceed from some definite organic lesion.

The most significant hemorrhages from the stomach arise from ulcer and cancer. Severe bleeding occurs if an ulcer opens into a considerable artery, though fatal hemorrhage is not very common from ulcer. The blood is often fresh and free from admixture with mucus or food. Repeated hematemeses may occur from a fissure or erosion so small as to be discoverable only with utmost care at operation, so that ulceration cannot be rejected as a cause without great circumspection. In general hematemeses serves to indicate gastric rather than duodenal ulcer, and melena has a contrary significance, but to a less degree.

The bleeding in cancer is rarely profuse, but in such small

amounts that it becomes partially digested and is ejected as "coffee-ground" material. Articles of food of a reddish color may simulate fresh blood in the vomitus, and bismuth, iron, and dark-colored ingesta may resemble the "coffee-ground" vomitus. The detection of red blood cells under the microscope or of blood coloring matter chemically or by the spectroscope may be necessary to clear the diagnosis.

### PUS

Pus is vomited after being swallowed, or as the result of suppurative gastritis or perigastric abscess from which it has reached the stomach. Subphrenic abscess, hepatic abscess, empyema, suppuration of bronchial glands and similar conditions are rare causes of purulent vomiting, the pus reaching either the stomach or esophagus through rupture of the wall.

### PARASITES

The round worm is not very infrequently vomited and the segments of tapeworm are occasionally seen. Very rarely the larvae of certain flies, trichinae, small echinococcus cysts from rupture of a larger cyst near the stomach, hookworms and possibly other parasites are ejected. Such occurrences should be investigated with a good deal of skepticism, since imperfect observation or deliberate deception is so often met with.

### FECAL VOMITING

This occurs as a result of intestinal obstruction from any cause, and earlier in proportion to the nearness of the obstruction to the pylorus, its acuteness and its completeness. It may be delayed indefinitely if the lesion be chronic and low in the intestinal tract.

After the stomach contents are ejected, bile and then the contents of the small bowel follow, becoming more and more offensive in most cases. If the vomitus in cancer of the stomach suddenly becomes fecal in character, without failure of bowel movement, a perforation of the lesion into a neighboring loop of bowel should be suspected.

### THE FECES

The normal adult passes some five or six ounces (150 to 180 gms.) of fecal matter daily, brownish from the presence of hydrobilirubin, but often modified by other coloring matter, of more or less semi-solid consistency, cylindrical in form, and of moderately offensive odor. The amount of fecal matter varies widely with habit as to diet and frequency of defecation. If concentrated foods, which are nearly all absorbed be used, but little matter is left to be passed through the stool. The total weight increases in certain types of diarrhea in which transudation of fluid into the intestine occurs.

### COLOR

The color of the stools is light in those using an exclusive milk diet, and dark in meat eaters. In diarrhea the color commonly lightens from the dilution with fluid. Iron, bismuth, and other medicines from which black sulphids are derived in the intestine give black stools. Purgation by calomel hurries bile along in the bowel with a resulting greenish or yellowish stool. Fresh blood gives a reddish stool and partially digested blood a dark, tarry appearance. In obstructive jaundice the color is pale, and if pancreatic secretion be absent the color is even lighter, from the abundant fat present.

The modifications in color from the use of blueberries, spinach, or other highly colored foods should be mentioned. The greenish stools in the infantile digestive diseases are colored by the growth of color-forming bacteria in the intestine.

### FORM

In the diarrheal diseases the stools become fluid, varying from the rice water stools of cholera to the mushy stools of ordinary mild looseness of the bowels. The former may show practically no fecal matter, while in the latter this is simply rendered softer and more dilute by increased fluidity. Very fluid stools are seen in mucous colitis, arsenical and antimonial poisoning, cholera nostras and cholera infantum.

Normal fecal matter is passed in cylindrical form and firm

enough to fairly maintain its shape. If the water of the liquid contents of the ileum be absorbed too completely in the lower bowel, we may have rounded masses of great firmness—scybala. They occur only when retention exists, being so formed by sacculi in the wall of the atonic bowel. They may contain lime salts in excess after long retention.

Pipe-stem and ribbon-shaped stools result from narrowing or deformity of the anus, from prolapse, stricture, spasm, cancer or other tumor, modifying the shape of the outlet of the bowel.

### REACTION

The stools of infants give an acid reaction, but may become alkaline in diarrhea. In adults it may be alkaline or slightly acid, being especially alkaline in typhoid fever.

### ODOR

The characteristic odor is due to indol and skatol, arising from putrefaction of proteid material in the colon. Hydrogen sulphid is occasionally present especially after use of saline laxative waters. The odor is marked during a meat diet, and very slight when milk only is taken. Fermentation of carbohydrate material gives a sour odor. Acholic stools are offensive because of decomposition which occurs in the absence of the bile. Gangrenous processes in the bowel, as in syphilitic, cancerous or dysenteric ulceration, give a more or less characteristic odor to the passages.

### ABNORMAL CONTENTS

Food remnants are especially abundant and unchanged in lien-teric diarrhea, but certain portions of a mixed diet may often be distinguished in the normal stool, especially if mastication has been imperfect. Milk curds often announce an attack of indigestion in the infant.

**Mucus.**—A small amount, well distributed, is not abnormal. If in larger masses it signifies an over action of the mucous glands of the large bowel. In mucous colitis it is present in masses, ribbons, or casts. In dysentery it is stained with blood. In general the abund-



ance of mucus in large masses points to colonic disease, while a lesser amount well distributed may have origin in the small bowel.

**Pus.**—If in large amount it comes from the rupture of an abscess into some part of the bowel, and a single discharge or separate purulent stools at intervals may be present. Pelvic abscesses in women and appendicular suppuration in either sex constitute the majority of such cases. Abscesses about the kidney, in the gall-bladder, liver and pancreas, subphrenic abscess, and empyema may also rupture into the bowel. Dysenteric and other ulcerative processes in the intestinal tract may cause the stools to contain pus for long periods.

**Blood.**—The normal appearance of the unmixed blood from the bowel signifies the occurrence of a fresh hemorrhage from the bowels, while the passing of "tarry" blood, retained for some time and mixed with the feces, is known as melena. In the latter case the bleeding has occurred high in the bowel, the blood has been mixed with the feces during its passage, and the color has been changed by the digestive processes. Bleeding occurs from conditions causing laceration or ulceration of the mucous lining of the digestive tract, from other conditions causing engorgement in certain constitutional diseases, and from rupture through the coats of the stomach or bowel from without. Swallowed blood may also, of course, discolor the stools. The laceration may be through external violence or foreign bodies in the bowel. The mucosa may bleed from the bites of the hookworm. Ulceration occurs in the form of peptic ulcer, gastric or duodenal, and in dysenteric, tuberculous, cancerous, syphilitic and typhoidal processes.

Engorgement of the intestinal mucosa occurs in conditions blocking the portal circulation, notably in cirrhosis of the liver and thrombosis of the portal vein. In thrombosis of the superior mesenteric vein and embolism of the corresponding artery bleeding into the bowel may be noted. In any of the purpuric diseases, leukemia, and hemophilia, bleeding may occur.

The choked condition of the incarcerated loop of the bowel accounts for the bloody stools of intussusception. The rupture of an engorged hemorrhoidal vein is the source of the blood in bleeding piles, the most frequent form of hemorrhage from the bowel.

Rupture of an aneurism into the digestive tract may cause a very

abundant and even fatal hemorrhage. The passage of a large quantity of dark blood would lead one to think especially of duodenal ulcer, and of fresh blood, of typhoid fever. Death may occur in either condition before the blood passes the anus, the symptoms being those of internal hemorrhage. The finding of occult blood in very small amounts from gastric ulcer or cancer, or similar lesions below, is an important factor in diagnosis.

**Fat.**—Fatty stools occur when the normal entrance of the bile and pancreatic juice into the bowel is prevented, and the fat thus remains undigested. The whiter stools characterize more especially pancreatic disease, and may consist mostly of fat. Glycosuria and jaundice are frequently present.

**Gall-stones.**—Gall-stones may be found after a so-called “successful” attack of biliary colic, the stools being mixed with water and passed through a sieve to facilitate the process. A faceted stone indicates that more than one has been present. The passage of many stones in a single stool justifies the diagnosis of a rupture of the gall-bladder into the bowel. One hundred and seventy-five thus passed at once with full recovery in one of my patients. The stone is found in but a very small percentage of cases after biliary colic, since it fails to pass the duct in most of these attacks. Many patients bring either particles of intestinal sand, seeds of fruit or other foreign bodies, or the pale greenish lumps of soapy appearance seen after taking the olive-oil treatment for gall-stone disease, believing them to be gall-stones. Pancreatic calculi consisting chiefly of carbonate of lime, are so friable that they are rarely recognized when passed.

**Intestinal Sand.**—Small grains of mineral matter, chiefly salts of calcium, are passed by certain patients suffering from secretory neuroses affecting the bowel. Vegetable concretions should be distinguished. They are often derived from bananas and pears, but may originate from other fruits and vegetables.

**Sloughs.**—These may occur as the result of the separation of the necrosed portion of the typhoidal patch, of the loop of the bowel in intussusception, of the pancreas in pancreatitis, of a polyp, or a patch of mucous membrane in dysentery, or of a cancerous growth.

**Enteroliths.**—Stony masses may result from the calcification of

long retained scybalous masses. Calculi made up largely of magnesium and calcium salts are rarely present. Bismuth and other drugs may form enteroliths, and even cause intestinal obstruction. Hulls of oatmeal, those of the core of the apple, seeds of various kinds, toothbrush bristles, hair, etc., may be found in the concretion.

**Foreign Bodies.**—Coins, pieces of bone, and various other substances swallowed by accident and especially swallowed by dime museum freaks and the insane, are occasionally found in the stools. Articles inserted into the bowel may be found. The Murphy button is occasionally a welcome constituent of the fecal movement.

**Parasites.**—The round worm, tapeworm, pin-worm, whip-worm, hookworm, and occasionally other parasites may be present in the stools. The ova are very frequently found. The examination of the stools for parasites and their ova is assuming greater importance in America since the acquisition of the tropical possessions renders the parasitic intestinal diseases so much more frequent here.

### MICROSCOPICAL EXAMINATION

Much interesting work has been done upon the feces resulting from certain standard diets. The conclusions of importance in diagnosis are summarized below.

If the fluid stool or a portion of firmer consistency disintegrated with water be placed under the microscope, one may see epithelial cells, squamous, and cylindrical, leukocytes, muscle fibers, ammonio-magnesium phosphate crystals, various forms of plant cells and enormous numbers of bacteria. Red blood cells may be recognizable, but the chemical test is much more reliable. Yellowish-brown muscle fibers are easily seen under the low power, and if but a few are seen to the field, more or less disintegrated, and with indistinct striae and nuclei, they are normal. In pancreatic disease especially, the fibers are abundant, and show little effect of the digestive process. Abundant connective tissue remnants point toward a weakened gastric digestion. Very abundant fat drops, crystals of the fatty acids, and of soaps are abnormal, and point to biliary obstruction or pancreatic disease. Fifty per cent. to 80 per cent. of the fat eaten may appear in the stool instead of the normal 5 per cent. to 10 per cent.

Vegetable remnants are much more abundant in the case of those vegetables eaten raw. Well preserved starch granules in abundance are an indication of hyperacidity, or of fermentative dyspepsia. They may be stained blue by Lugol's solution, the color assuming a reddish tint if erythrodextrin be present.

Fragments of malignant growths are occasionally found, but the histological examination should be very complete before basing a diagnosis upon it.

The ova of intestinal parasites are sought after thorough disintegration of the feces, and if present afford positive evidence of infection. The range in diameter generally lies between 30 and 100 microns, and they may usually be recognized by a comparison with the illustrations given.

The *Entamoeba histolytica* must be distinguished from the ameba of Loesch, which is non-pathogenic and found by Craig in 65 per cent. of normal individuals. The *Trichomonas intestinalis*, *Cercomonas hominis* and *Megastoma entericum* are common flagellate organisms. Many other parasites, chiefly non-pathogenic so far as we now know, may be present.

The colon bacillus is a normal inhabitant of the bowel and is generally harmless. Most of the bacteria present in the feces are saprophytic, and they seem to be essential to proper intestinal function. The important pathogenic organisms are those of typhoid, tuberculosis, cholera and bacillary dysentery.

#### CHEMICAL EXAMINATION

The most important feature of the chemical examination of feces is the detection of blood. Care must be taken that no extraneous blood be introduced, as from the vagina, from hemorrhoids or even from meat in the diet. The guaiac test is given by Webster as follows:

"A small portion of the stool is rubbed up with water and one-third of its volume of glacial acetic acid added. This mixture is well shaken in a test-tube and a few cubic centimeters of ether added. After thoroughly shaking this mixture, it is allowed to settle, when the ether, in the presence of blood, will have assumed a brownish

color. In case the ethereal extract is not clear, a few drops of alcohol may be added. On adding to this ethereal extract a mixture consisting of equal parts of fresh tincture of guaiac and ozonized turpentine, a blue ring will form at the point of contact or a blue coloration will be seen throughout the mixture if the tube be shaken."<sup>1</sup>

The aloin test is far more delicate but requires especial care in the exclusion of foods containing hemoglobin.

For a more detailed study of the feces the reader is referred to the special works upon the subject.

## THE URINE

### CLINICAL EXAMINATION

The urine for clinical investigation may consist of a single specimen for the qualitative examination, or a sample of the mixed 24-hour urine for the more exact quantitative estimation. The variations in quantity from changes from warm to cold weather, because of free perspiration, diarrhea, increased intake of fluid and nervous conditions accompanied by over-activity of the kidneys, should be considered. The secretion is relatively greater for the body weight in children than in adults. The average quantity as given by many observers is in the general neighborhood of 1500 c.c. with very wide physiological limits of variation. The total amount is increased in diabetes of both forms, in chronic interstitial nephritis, in amyloid disease of the kidneys, in hysteria, and many other nervous affections, and during the absorption of large effusions. Oliguria results from a poor circulation through the kidneys, as in cardiac disease, especially when resulting in low vascular pressure, in obstructive types of nephritis, in passive congestion of the kidneys, and under conditions in which fluid has been lost from the system, as in diarrhea, hemorrhages, and severe vomiting, or has not been absorbed in proper amount, as in high intestinal obstruction and acute dilatation of the stomach.

The specific gravity varies in single specimens from 1.001 or

<sup>1</sup> Webster: "Diagnostic Methods."

1.002, as after drinking much beer, to 1.040 in urine of fever or 1.070 in diabetes, but the average in health for the 24-hour specimen lies between 1.015 and 1.025. Dr. T. R. Love found the average of a long series of examinations in my laboratory to be slightly over 1.020 after excluding all cases of diabetes, probably higher than the average in moister climates.

**Color.**—The normal amber color is due to urochrome, uroerythrin and urobilin in varying proportions. The color becomes lighter as the quantity increases, and generally as the specific gravity decreases. In diabetes mellitus, however, the color is pale because of the increased quantity of urine, yet the specific gravity is high because of the sugar contained. A greenish tinge is often present. The urine is abnormally colored by various drugs, and through the changes induced in the system by various diseases. Carbolic acid renders it darker. Methylene blue gives a bluish-green color, and santonin, chrysarobin, iodin, senna and rhubarb give a yellow color, changing to red upon the addition of an alkali.

Bile pigment gives a yellowish, brownish or almost black color, with yellowish foam upon shaking. Fresh blood renders the urine red, while the dissolved pigment gives a reddish brown, coffee-colored or black shade. The reddish color is due rather to the hemoglobin contained and the darker color to methemoglobin. A dark red color may appear if hematoporphyrin be present as after the use of sulphonal and certain other hypnotics. An almost black urine is occasionally passed in melanotic disease, but the color may appear only upon standing. Alkaptonuria and ochronosis may give a similar discoloration. The alkaptan reduces Fehling's solution, while the melanin-containing urine does not.

A dark-colored urine is secreted in cases of intestinal putrefaction, peritonitis, gangrene and suppuration, due to indican in excess, or to certain phenol derivatives.

**Transparency.**—Normal urine is clear excepting for the slight cloud of mucus that settles upon standing. If alkaline, as after a hearty meal, the precipitated phosphates render it cloudy. Upon cooling, urates settle to the bottom, and crystals of uric acid may be found. A turbidity with a peculiar shimmer when held toward the

light is seen in urines heavily infected with colon bacilli and other organisms. Pus, blood, epithelial cells, chyle and even milk added for the purposes of deception may render the urine turbid or completely destroy its transparency.

**Odor.**—The normal aromatic odor may be replaced by a foul ammoniacal smell if the urine be decomposed by bacterial infection, as in cystitis, this constituting the most important departure from the normal. The fruity odor of acetone present in diabetes may pervade the whole room if the urine has stained the bedding. Asparagus, turpentine, cubebs, menthol, copaiba, valerian, asafetida and other substances give more or less characteristic odors.

**Reaction.**—The normal urine is acid chiefly from the presence of acid salts, especially  $\text{NaH}^2\text{PO}^4$ . The total acidity of the 24-hour specimen is roughly equivalent to that of one or two gms. of HCl. The taking of mineral acids and meat diet increases the acidity, while the use of a vegetable diet, rich in alkaline substances, renders the urine temporarily alkaline. Persistent alkalinity is generally due to decomposition in the bladder due to micro-organisms, as in most cases of cystitis. In general infection with the colon bacillus, the urine may be turbid and the odor extremely foul and yet the reaction be acid to litmus paper. The giving of organic acids, especially citric and tartaric, in moderate dosage does not render the urine acid, but because of their being oxidized in the system to carbonic acid which combines to form basic salts, a lessened acidity or even alkalinity results. A strongly acid urine is commonly found in gout, rheumatism, many febrile diseases and in digestive disturbances with decreased gastric acidity.

**Urinary Solids.**—The most important function of the kidneys being the elimination of the urinary solids, attention should be given to their clinical estimation in many cases. The normal amount is 60 to 70 gms., and a constant diminution from the standard is significant of grave disorder of excretion.

If sugar be excluded we may get a fairly accurate estimation of the function of the kidneys as regards the excretion of urinary solids by multiplying the last two figures of the specific gravity of the 24-hour specimen by the coefficient 2.33, this giving the quantity in 1000

c.c. Thus if the specific gravity be 1.020 and the total amount 1500 c.c. we have 20 times 2.33, which equals 46.60 gms., representing 1000 c.c. of urine. Adding  $\frac{1}{2}$  because of the 500 c.c. in excess of the 1000 estimated we have a total of 69.90 gms., a normal amount for a large healthy adult.

A close approximation may also be reached by multiplying the last two figures of the specific gravity by the number of ounces in 24 hours and the product by 1.1, the result being in grains. The example given above would indicate by this method: 20 times 50 times 1.1 equals 1100 grains, or slightly over 70 gms.

The decreased elimination of the solids may be due to disease of the secreting structure of the kidney or to disease elsewhere, and is sufficiently serious to demand investigation.

**Pneumaturia.**—The passage of gas or air from the bladder may occur because of the fermentation of the urine, communication between some air or gas containing viscus with the bladder, or because air has been introduced in a cystoscopic or other examination. Most instances are due to the presence of the colon bacillus in a urine containing sugar, carbonic acid being formed. The yeast fungus and the *Bacillus aërogenes capsulatus* are occasionally the causative organisms. The gas may bubble out of the catheter if the end be placed under water.

### CHEMICAL EXAMINATION

The chemical and microscopic investigations of the urine are described in the special works upon the subject, and we shall deal only with the clinical significance of the findings.

**Chlorids.**—An increase of the total chlorids beyond the normal 10 or 15 gms. is often noted in recovery from pneumonia, when large effusions are being absorbed, and in diabetes insipidus. A marked decrease is to be found in starvation, in the exudative stage of acute pneumonia, in most fevers, during the increasing stage of large effusions and in many serious digestive diseases.

**Phosphates.**—The decrease from the normal 2 or 3 gms. noted in pregnancy, nephritis, gout and certain other conditions is not of great clinical importance. An increase is often noted in serious nervous



disease and in dyspeptics. In the so-called "phosphatic diabetes" increased thirst, loss of flesh and debility coincide with an increased excretion of phosphates.

**Sulphates.**—**INDICAN.**—Since the sulphates are chiefly derived from the breaking up of albuminous substances in the body their excretion increases in acute and chronic wasting diseases. Uniting with indoxyl formed in intestinal putrefaction from indol, the conjugate potassium indoxyl sulphate, or indican, is formed. Thus indican becomes an important measure of the amount of intestinal putrefaction. This occurring more freely in the absence of HCl in the gastric juice, indican in excess is somewhat significant, in certain cases, of the presence of gastric cancer. In others it points toward obstruction of the small intestine.

**Oxalates.**—A few milligrams of oxalic acid are excreted daily, chiefly as calcic oxalate. This is increased in many cases in which carbohydrate fermentation occurs, and may be found after eating rhubarb, tomatoes and certain acid fruits. Its chief importance lies in the irritation of the sharp crystals as they pass through the urinary passages, the diagnosis of renal calculus being often wrongly made in these cases. If retained, calcic oxalate calculi may form. The neurasthenic and other nervous disturbances seen in these patients are due rather to the indigestion and faulty metabolism than to the formation of the crystals.

**Urea.**—Most of the nitrogen eliminated from the body is in the form of urea, the amount averaging about 30 gms. It is derived from the consumption of body tissues and the proteids of the food. Thus it varies greatly according to the stress which the body undergoes and the character of the diet. In fevers and wasting diseases the amount derived from the body waste is much increased. In diabetes this amount is still further increased by the abnormally large intake of food.

It is decreased in conditions affecting the capacity of the liver to form it, as in cancer, and in conditions of the kidney preventing its elimination as in nephritis. Uremia is common in those patients who do not excrete a normal quantity of urea, for the very conditions which interfere with its excretion prevent the elimination of other

poisons which are found in the system in uremia. The severity of the nephritis may be fairly gauged by the ability of the kidneys to excrete urea as shown by quantitative estimates or indirectly by the estimation of the total solids.

**Uric Acid.**—This product of the decomposition of nucleoproteids is excreted in a quantity of a gram a day or less, and should be found in a ratio approximately of 1:50 to urea. Its clinical importance has been greatly overrated in the past. It is increased under the conditions causing increase of urea and also in diseases accompanied by a heavy leukocytosis, and most of all in leukemia. The relationship of uric acid to gout and lithemia is still under discussion. It is increased during the attack of gout according to Fletcher, but decreased before and after. Whether it causes the symptoms of lithemia is an open question, there being no such conclusive evidence as the deposit of crystals of the urate of soda about the joints in gout affords. An excess of sodium salts in the lymph and synovial tissues is probably an essential factor in the precipitation of the uric acid in this disease.

For all details regarding the excretion of ammonia, the xanthin bases, melanin, phenol and other substances of more interest to the physiologist than the clinician, the special works upon urinary chemistry should be consulted.

**Albumin.**—This term used without qualification refers to serum albumin, by far the most important member of the group to the clinician. With it are often associated one or more of the other proteids of the urine, serum-globulin, albumose, nucleo-albumin, fibrin, etc. We may assume that the presence of albumin in the urine signifies some structural or functional change in the kidney, but the very fact that the term physiological albuminuria is so commonly used implies that albumin may pass through the epithelial cells of the tubules and the glomeruli when they are so little compromised that no demonstrable post mortem changes can be found. Probably transient albuminuria is associated with mild circulatory changes in the cells, and various grades of pathological change exist up to the extreme lesions of chronic nephritis.

In general, less importance is attached to the finding of bare

traces of albumin in the urine than was the case a few years ago. With the refinement of chemical methods it is easy to find at times traces of some of the albuminous bodies in many urines which are substantially normal so far as the physician's point of view is concerned, since other samples of urine from the same patient show nothing abnormal. The more or less constant presence of albumin, however, raises the presumption of organic disease.

**ACCIDENTAL ALBUMINURIA.**—We may find albumin in the urine which comes from an extrarenal source and hence affords no grounds for the diagnosis of renal disease. After the urine escapes from the tubules of the kidney it may become contaminated with blood, pus, chyle, semen, leucorrheal discharge, the products of inflammation in pyelitis, cystitis, prostatitis, vesiculitis or other similar conditions. In men, the cleansing of the urethra by the first urine passed renders this specimen most likely to contain urethral, prostatic or vesicular secretion, and hence the middle urine is a fairer sample. Pus from the bottom of the bladder may show more prominently in the last portion passed. The microscopic examination of the sediment may show vaginal epithelial cells, bladder cells, blood clots or other evidence pointing toward an extrarenal origin of the albumin, while the tube casts and renal epithelial cells will indicate that part of the albumin at least is of renal origin.

**FUNCTIONAL ALBUMINURIA.**—Those cases in which albumin is found in the urine after severe exercise, after being long in the upright posture (orthostatic albuminuria), after a cold bath or a hearty meal, or intermittently, in persons otherwise in good health and showing no sedimentary or other evidence of renal disease, may be considered as functional. We may at least state that no organic changes are recognized in the kidneys in such cases. Certain types of albuminuria in pregnancy and in adolescence are perhaps to be assigned to this class. The life insurance companies are probably right in declining to insure applicants from this class of patients, since certain individuals may develop the signs of definite nephritis after years of very slight and intermittent albuminuria, and it is impossible to tell in advance which ones will escape.

**ALIMENTARY ALBUMINURIA.**—It is well recognized that certain

individuals may pass albumin in the urine after eating several eggs or excess of other albuminous food. These cases may well be regarded with suspicion.

**TOXIC ALBUMINURIA.**—The circulation through the kidneys of blood containing substances irritating to the epithelial cells results in the escape of albumin through them. The poison may come from without, as from an anesthetic or vapor of turpentine inhaled, or from a poison swallowed, as cantharides, corrosive sublimate, lead, carbolic acid, etc., or introduced by inunction, or injection by rectum or vagina, or hypodermically; or from a poison to the cells derived from some pathological process in the body, as in the purpuric diseases, severe anemias, and the leukemias.

**FEBRILE ALBUMINURIA.**—This probably results from damage to the cells by substances produced in the febrile paroxysm and is therefore ultimately of toxic origin. The cellular degeneration of prolonged fever must be considered apart from the toxic cause. In typhoid fever and most of the other acute infectious diseases, albuminuria appears during the height of the fever and is more abundant in accordance with the severity of the infection.

**ALBUMINURIA OF PASSIVE CONGESTION.**—When the return blood flow is interfered with, as in heart disease, advanced emphysema and those conditions producing pressure upon or obstruction of the renal veins, the engorgement of the kidney results in the passage of albumin into the urine. The albuminuria may be proved to be unilateral by segregation of the urine in these cases. In floating kidney torsion of the pedicle or kinking of the renal veins occurs, and as these conditions are more prominent in the upright posture, certain cases of the orthostatic albuminuria are of this origin.

**NEUROTIC ALBUMINURIA.**—In many nervous diseases albumin may be present in traces, without organic kidney disease, as in epilepsy, exophthalmic goiter, apoplexy, migraine, etc.

**ALBUMIN OF ORGANIC DISEASE.**—Albumin is present in pyelitis, derived chiefly from the pus secreted, and it may also be present when abscess in or around the kidney gains access to the pelvis or ureter. In acute nephritis the large amount of albumin is augmented by that in the exuded blood. In chronic interstitial neph-

ritis, in amyloid disease and in congenital cystic kidney, the amount is generally small. In chronic parenchymatous nephritis it may be sufficient to cause the urine to "boil solid," approximately 2 per cent. of dried albumin by weight being requisite. In tumors of the kidney, notably in hypernephroma, albumin is often present, and may be derived from the blood which is commonly noted.

The presence of albumin by no means justifies the diagnosis of Bright's disease, nor does its absence justify the exclusion of nephritis. In well marked interstitial nephritis albumin may be absent at half the examinations for a time, and present in the merest traces at the others. In this form the other signs of the disease are of special importance in enabling us to avoid error.

**Serum Globulin.**—This has practically the same significance as serum albumin, with which it is commonly found. An excess of the former over the latter is said by Senator to characterize amyloid disease of the kidney.

**Nucleo-albumin.**—This form is believed to be indicative of catarrhal and desquamative processes in the bladder or other portions of the urinary tract. Many urines reported to contain a bare trace of albumin, especially in slight illnesses, probably have this form only, and it possesses but little clinical significance in such cases. Traces of nucleo-albumin are even thought to be physiological by some authorities.

**Bence-Jones Protein.**—The presence of the Bence-Jones body in considerable quantity should lead to the examination of the patient for multiple myelomata, to which it points with considerable certainty. It has been found in lymphatic leukemia.

**Albumose.**—This body is of less definite significance than other albuminous substances, since "it is due to the breaking down of a tissue or exudate, and may therefore appear in almost any type of disease."—(Webster). It is an interesting finding in many suppurative, febrile, digestive and hepatic disorders in which hemolysis occurs.

**Blood.**—We may find a few scattered red cells in the urine, or a general red color from a greater admixture, or definite clots may be passed from the bladder. The urine becomes darker in color

from the hemoglobin dissolved from the red cells or the methemoglobin derived from it.

The red cells may appear normal under the microscope, or if long exposed to the dissolving influence of the urine upon the hemoglobin, may appear as "shadows" only. The cells become crenated in a concentrated urine. They may be separate, or massed together to form clots, or adherent to casts and epithelial cells. Blood casts are especially characteristic of acute nephritis.

**SOURCE OF THE BLOOD.**—Hemorrhage of the kidney is seen in acute congestion, such as results from the passing of cantharides, phenol and other irritants through the cells, from the malignant types of the acute infectious diseases, in the purpuric group and in hemophilia, and from malaria; in acute nephritis constantly and in large amount, and in chronic nephritis, more sparingly or not at all for a time. The hemorrhagic type of chronic interstitial nephritis is probably more common than most physicians believe, and may be unilateral and of extreme severity. In one such case the hemoglobin was reduced to 40 per cent. but returned to normal after decapsulation of the affected kidney, although the albumin and casts have persisted.

In tuberculosis, cancer, aneurism, filariasis, distomiasis and after renal infection hemorrhage occurs. One of the most important types is that caused by stone in the pelvis of the kidney. Traces of blood frequently appear in the urine after the handling to which a floating kidney is subjected in the course of a physical examination, and its amount may be taken as a gauge of the sensitiveness of the displaced organ. I believe only an engorged organ will bleed after the very moderate traumatism of the physical examination. In the so-called renal epistaxis the blood doubtless comes from the kidney, but the exact pathology is unknown. The bleeding from hypernephroma is of especial importance in the diagnosis in this type of tumor. From the ureter blood may be passed as the result of the irritation of a stone, or crystalline sediment, especially calcic oxalate.

Hemorrhage from the bladder occurs as the result of a severe cystitis, of papillary and cancerous tumors, of tuberculosis, from certain parasites, especially the *Bilharzia hematobia*, from simple

ulcer, rupture of a vein, in chyluria, and especially from the irritation of vesical calculus. Trauma is a not infrequent cause.

From the urethra we may have blood as a result of instrumental or other form of traumatism, from impacted calculus, from ulceration or tumorous growth, and in the acute inflammatory stage of gonorrhea. The dark color of the so-called "black clap" is the result of such hemorrhage.

If the bleeding be from the kidney substance the individual cells are found thoroughly mixed with the urine, paler than normal or oftentimes massed together as blood casts. The urine is darker than normal from the dissolved blood coloring matter and from the admixture of red cells. The presence of other casts and of renal epithelium adds to the probability that the hemorrhage comes from the kidney. A large amount of albumin points to a renal source. If from the pelvis casts should be absent, while clots of irregular shape from the pelvis or the long slender ones from the ureter may be found. If the pelvic hemorrhage be from stone abundant caudate cells are found. Since the tailed cells are also found in the bladder, the diagnosis is to be made from the association of the definite signs and symptoms, and by no means from the cells alone. Ureteral hemorrhage can scarcely be differentiated from that from the pelvis.

A fresher appearance of the blood, the occurrence of larger and more irregular clots, a less perfect mixing of urine and blood, the occurrence of bladder epithelial cells, especially in cases of cystic stone, the presence of mucus in many cases, and the absence of casts, signify that the blood comes from the bladder. Clots too large to have passed through the ureter may be found. The blood cells are generally little altered. The two-glass test shows more blood in the last specimen.

From the urethra the blood may flow perfectly fresh since no admixture with the urine has occurred. The channel is washed out upon passing urine so that the last passed urine is entirely clear. In case of doubt as to the origin of the blood in the urine the cystoscope and the ureteral catheter generally suffice for an exact diagnosis. Blood from extra-urinary sources may be found, but it is

easily detected if the possibility be kept in mind. Prostatic hemorrhage is common enough in old men to deserve mention.

**HEMOGLOBINURIA AND METHEMOGLOBINURIA.**—The blood pigment found in the urine may be hemoglobin, but it is more commonly methemoglobin, this being an oxidation product of hemoglobin. Even if the hemoglobin be present when the urine is passed, methemoglobinuria appears if the acid urine stands for some time.

These pigments appear in the blood when the normal capacity of the liver to convert the colored matter of the red cells destroyed in the body into bilirubin is overtaxed, and the blood then frees itself of the superfluous pigment through the urine. The liberation of 1 per cent. or 2 per cent. of the hemoglobin of the body is probably sufficient to cause hemoglobinuria.

Hemoglobin is found in the urine as the result of the action of hemolytic poisons, of which the most commonly met with are chloroform, carbon monoxid, phenol, the chlorates, arseniuretted hydrogen, phosphorus and snake poison. Transfusion of blood may be a cause. In grave infectious diseases, such as typhoid and yellow fevers, scarlet fever, variola and syphilis, and in severe types of malarial fever, it is often seen. "Blackwater fever" received its name from the darkening of the urine because of the presence of the blood cells and hemoglobin set free through the effects of the malarial poison. It is rarely seen in the tertian and quartan infections of America, and is probably almost exclusively due to the æstivo-autumnal type.

The paroxysmal form of hemoglobinuria is seen typically after exposure to cold and after muscular exertion. Chill, fever, backache and malaise precede the onset. The attack passes off after a day or two. The frequent association with Raynaud's disease and with syphilis deserves mention. The character of the hemolytic toxin in paroxysmal hemoglobinuria is unknown, as it is in the epidemic type seen in the newborn.

**Hematoporphyrinuria.**—This has become more important in recent years because of its occasional occurrence in those using sulphonal, trional and tetronal habitually. The port wine color is due to an iron-free derivative of hemoglobin. No especial diagnostic signifi-



cance attaches to the condition when occurring in tuberculosis, Addison's disease, syphilis and the acute infectious diseases.

**Alkaptonuria.**—This is of importance because of the fact that Fehling's test for glucose gives a positive reaction, but with an almost black color. The darkening of the clear urine upon standing or upon the addition of an alkali is characteristic.

**Glycosuria.**—When the normal one part per thousand of glucose in the blood is increased materially, glucose appears in the urine. In the alimentary form it is a temporary condition due to the effort of the body to rid itself of a hyperglycemia, resulting from a too great intake of starch and sugar. Glycosuria may also occur temporarily after acute fevers in stout persons and in certain nervous diseases. In suffocation, ether and chloroform narcosis, and other conditions resulting in a diminution of the normal oxygen supply to the system, glycosuria often develops. Its constant occurrence over long intervals is characteristic of diabetes mellitus.

If phloridzin be given sugar may appear in the urine even though not in excess in the blood, because of the changes induced in the renal epithelial cells. The diabetic temporarily free from glycosuria may be detected by giving 100 gms. of glucose or cane sugar upon an empty stomach, his lowered limit of tolerance being shown by the appearance of glucose in the urine in an hour or two. A similar test of greater significance consists in finding such a glycosuria after the ingestion of starchy food only.

The discussion of the influence of pancreatic, hepatic, and nervous diseases in the production of diabetes is out of place in a work of this character.

Sugar is so often falsely reported in urines submitted for examination because of the reducing action of uric acid, albumin, bile pigments, and many other substances that the student should be cautioned not to accept the result of any of the ordinary tests for glucose without confirming the findings by carefully executed fermentation tests or other conclusive procedure. The polariscopic test is suitable if available.

**Other Sugars.**—Lactose is found in the urine of nursing women, and may be present with glucose in diabetes. Levulose may appear

after eating fruit rich in fruit sugar, or in diabetes. Dextrin in the urine is of little clinical significance. Pentose probably gives rise to the osazone which causes Cammidge's reaction in pancreatic disease when the urine is treated with phenylhydrazin. This test is not at present regarded as pathognomonic of disease of the pancreas.

**Acetone.**—An increase over the normal trace of acetone found in the urine is present in conditions of poor nutrition, as in many fevers, chronic digestive disease, and after chloroform anesthesia. When the carbohydrates of the diet are decreased it may increase greatly, and is of especial significance as a danger sign in diabetes. Its presence calls for less strict exclusion of carbohydrates from the diet, and coma is to be feared if acetone continues in spite of treatment.

**Diabetic Acid.**—This is generally found in diabetic urine in conjunction with acetone, and adds to the bad prognostic import of the latter in this disease. Fatal acidosis is to be regarded as imminent if the amount be large. The presence of oxybutyric acid in addition to the two substances mentioned renders the outlook still more ominous.

**Diazo-reaction.**—This test, introduced by Ehrlich, has been a great disappointment to the clinician, since it is almost valueless as a differential sign between certain diseases which are confused in their early stages. Thus it is generally present in typhoid and in general acute miliary tuberculosis and tuberculous meningitis, and is of value only by its continued negative result, thus rendering these conditions improbable. Its presence in pulmonary tuberculosis indicates a grave course of the disease. Its presence in many other diseases is of little value, since the differentiation must be made entirely upon other signs and symptoms.

**Lipuria and Lipaciduria.**—These conditions, representing the presence of fat or of fatty acids in the urine, are pathological.

Fat is present with blood cells, albumin, etc., in chyluria, rendering the urine milky in appearance. Search should be made for the *Filaria sanguinis hominis* generally present.

Microscopically fat is present in chronic parenchymatous nephri-

tis, being found free or in the degenerated epithelial cells or epithelial casts. In late stages of acute nephritis it may be found. In diabetes, after excessive feeding of cod liver oil or other fats, after fracture and resulting fat embolism, and in bone diseases in which the marrow is affected, fat is often present. It should not be considered of pathological significance unless attached to cells or casts, until the possibility of its having come from oil used on the catheter or other extraneous source is excluded.

Lipaciduria, in which formic, butyric, and other fatty acids are present in the urine, is occasionally seen in diabetes and other diseases, but has no especial clinical significance.

**Cryoscopy.**—The determination of the freezing point of the urine and estimation therefrom of its molecular concentration has not proved to be of sufficient clinical value to call for discussion.

#### MICROSCOPICAL EXAMINATION OF THE SEDIMENT

No examination of the urine is at all complete without the microscopical examination of the sediment. Such changes occur in the crystalline sediment, after the alkaline decomposition of the urine, and such destruction of the cellular elements of the sediment, that a fresh specimen (drawn by the catheter in women if the examination is of particular importance), preserved by the addition of a drop of formalin, should be obtained. If the urine is alkaline when passed, as in cystitis, it should be examined as speedily as possible. Many mistakes have been made by examining a specimen from the wrong patient, or contaminating the sediment of one urine with the pathological findings of another through the use of unclean pipettes, etc. These matters deserve especial attention in case operative intervention is being considered. In women the catheter should be used with extreme care not to soil its tip by a false entry, since the urethral orifice may have been scrupulously cleansed, yet the urine be contaminated by the error of inserting the catheter into the urethra after it has been soiled by a mistaken insertion into the vagina.

**Blood.**—The presence of blood has already been considered under the title of hematuria.

**Leukocytes.**—A few white cells in the sediment are to be found

normally. Their nuclei are more distinct if the urine be acid or if it be rendered so by acetic acid. They may be stained in various ways if desirable.

**Pus.**—Pyuria is common as the result of formation of pus within the urinary tract or from contamination of the urine by pus from without. Albumin is found if any notable amount of pus is present.

The pus cells from the urethra in urethritis may be squeezed out or may pass out with the first urinary stream, so that they are easily classified as to origin. If the urethritis be confined to the anterior portion of the tube the first portion of urine is purulent, the second clear. If the posterior urethra be also involved, both portions are likely to contain pus. Suppurating lesions connecting with the urethra must be borne in mind.

In cystitis the urine is commonly alkaline, contains much pus, and a great number of epithelial cells. The alkaline reaction causes the pus to be "stringy." Blood cells are especially common if stone be the cause of the cystitis. The last portion of the urine contains most of the pus; the first portion may wash out the pus of a coincident urethritis, a clear stream following for an instant, before the pus of bladder origin appears.

Simple or malignant ulcer of the wall of the bladder, an impacted stone, a diverticulum which does not drain well, or irritation of the bladder wall by an abscess or growth in contact with it may cause pus to appear in the urine.

The obscured nuclei of the pus cells in alkaline urine may be cleared up by the addition of acetic acid. Most of the cells are of the ordinary polymorphonuclear type.

Pus from the ureter does not differ from that from the pelvis of the kidney, and the condition is to be determined by the catheterization of the ureters.

**PYELITIS.** From the pelvis of the kidney we often have pus in pyelitis, pyelonephritis, and abscesses breaking into the kidney. The constant appearance of pus in acid urine, in moderate amounts, and generally accompanied by a few erythrocytes, is characteristic of renal tuberculosis. A similar finding may be noted in chronic infective pyelitis, as after typhoid, and in cases of stone in the pelvis of

the kidney. Since the pus-containing urine irritates the bladder, frequent micturition often attracts the physician's attention, and a diagnosis of cystitis is wrongly made, or, if the cystitis be present, the antecedent pyelitis is overlooked. The detection of tubercle bacilli in the urine or the positive result upon inoculating a guinea pig, may be necessary to establish a diagnosis. More pain and hemorrhage are found in pelvic stone as a rule, although an encysted pelvic stone may give but little pain. Infectious pyelitis may exist for years without marked symptoms provided the drainage be good, yet the diagnosis is easily made upon microscopical examination of the urine. An appreciable proportion of applicants rejected for insurance because of albuminuria, owe the condition to an old pyelitis absolutely devoid of subjective symptoms.

Pus may suddenly appear in large quantities in the urine from one kidney, because of the rupture of an abscess into the pelvis or ureter, or because the ureter has been blocked for a time by kinking or by a shred of pus or clot of blood. One kidney may secrete normal urine and if the diseased one be blocked nothing may attract the examiner's attention, yet the next urine may be loaded with pus and bacteria. Much judgment is required to avoid an error in diagnosis in certain cases of this general type.

From the kidney substance itself but little pus is given off excepting in cases of frank suppuration. Round renal cells are present, and the pus is likely to form the so-called "pus casts." Casts of other varieties may have a few pus cells adherent. The presence of great numbers of pus cells should raise a presumption that some other part of the urinary tract is affected. The pus cells may be grouped together in irregular masses, especially if coming from an abscess. The presence of shreds covered with or made up of pus and epithelial cells, and generally easily seen with the naked eye, points to an antecedent posterior urethritis (the so-called "gonorrheal threads"). The presence of degenerated pus cells, dead spermatozoa, and epithelial cells, points to a chronic vesiculitis, and such material is often found at the meatus after the usual examination of the prostate and seminal vesicles.

Pus, blood, and shreds of tissue, fecal matter, and bacteria are

found in differing combinations in those cases in which appendical abscesses, extra-uterine pregnancies, suppurating cysts, malignant tumors, etc., have contaminated the urine. Each case must be worked out in accordance with the evidence.

**Spermatozoa.**—The presence of spermatozoa in the urine commonly indicates that they have been swept out of the urethra after a seminal emission by the first rush of urine. An over-distended seminal vesicle so easily discharges its contents through pressure of a constipated fecal movement that but little importance attaches to the finding of a few spermatozoa in the urine. Testicular casts are often passed with the spermatozoa. Semen may escape from the vagina after coitus and contaminate the female urine.

Fecal matter may be passed regularly in case of rectovesical fistula or other means of communication between the urinary passages and the bowel. Thread-worms may be passed in these cases. Fragments of malignant or other tumors, especially a vesical papilloma, may pass in the urine, and may be in sufficiently good state of preservation to be of value histologically.

**Parasites.**—The most important are the ova of the *Schistosoma hematobium*, and the embryos of the *Filaria sanguinis hominis*, both associated with hematuria, and the latter with chyluria. The *Trichomonas vaginalis*, *Anguillula acetæ*, amebæ, *echinococcus hooklets*, or fragments of a cyst wall, and rarely the *Strongylus gigas* have been found.

Calculi of small and moderate size may pass in the urine, and may come from the kidneys directly, from the bladder, or the urethra. The phosphatic and ammonium urate stones are generally of bladder origin, but may come from the urethra. Uric acid, calcium oxalate and the rare cystin, indigo, urostealith and Xanthin stones are formed in the kidney as a rule.

**Bacteria.**—Normal urine should be practically free from bacteria. The average specimen examined has been more or less contaminated before it reaches the stage of microscopic examination, and a few organisms may pass without attracting serious attention.

If there be a blood infection, especially by the typhoid or colon bacillus, myriads of bacteria are excreted through the kidney and

often give a distinct turbidity to the specimen—a condition termed bacilluria. Lesions in the course of the urinary tract may permit streptococci, staphylococci, gonococci, tubercle bacilli, ray fungi, etc., to infect the urine. The yeast fungus is common in diabetic urine. The smegma bacillus constitutes a common contamination and requires a special watchfulness in case the tubercle bacillus is to be sought. The micrococcus ureæ is present in ammoniacal urine. Bacteria of many varieties are found in infections of the urethra, bladder, and pelvis, for a full description of which the reader is referred to the special works on the subject.

**Crystalline Sediment.**—In acid urine we may find uric acid crystals, and calcic oxalate commonly, and rarely hippuric acid, cystin, leucin, tyrosin, and a few other crystallin substances. In concentrated urines an abundant precipitate of acid urates of sodium and potassium often occurs, giving the pinkish or reddish “brick dust” sediment. It is soluble upon boiling. In alkaline specimens the most common crystal is the familiar one of triple phosphate. With it we find associated dumb-bell crystals of calcium phosphate and calcium carbonate, plates of magnesium phosphate, “thorn-apple” crystals and needles of ammonium urate, amorphous phosphates, and carbonates of magnesium and calcium. Cholesterin plates are occasionally found, and the blue needle-shaped crystals of indigo.

**Epithelial Cells.**—A few irregular flat cells from the bladder and urethra are normally present in the sediment, and in the urine of women similar cells from the vaginal wall, generally in stratified groups as they have scaled off from the mucous surface. The presence of many cells of either form mentioned denotes a desquamative type of inflammation of the mucous membrane involved. Pus cells become more abundant as the inflammation progresses. Following the desquamation of the flat epithelial cells of the urethra, bladder, ureter, and pelvis of the kidney, there may be thrown off cuboidal and columnar cells from the deeper layers, the flat cells eventually being outnumbered by them as the superficial layers are removed by the progressive desquamation. The flat cells are granular with a central nucleus.

From the deeper layers of the ureteral mucous membrane and the

pelvis, "tailed" cells are given off. If present in great numbers they are generally held to indicate a pyelitis of some severity. This conclusion is probably justified if the associated symptoms point toward the kidney rather than the urethra or ureter as the seat of the disease. From the well-known frequency of pyelitis and the abundant desquamation present in it, in case of doubt, a pyelitis becomes the most probable diagnosis.

From the tubules of the kidney we have round cells, somewhat larger than leukocytes, with a relatively large nucleus. These are the ones so often adherent to tube casts in acute nephritis. Although showing but little change at first, they later show granular degeneration or definite globules of fat, according to the chronicity of the inflammation. The somewhat similar cells from the ureters and prostate are about twice the diameter of a pus cell, and may thus be differentiated from the smaller renal cells. A correct inference as to the origin of any of the cells found in the urine is possible only through a consideration of their size, shape, changes from the action of the urine after standing, their number, and especially the associated signs and symptoms of the case. The laboratory findings should be regarded only as supplementary to the clinical diagnosis.

**Tube Casts.**—For the recognition of renal diseases we depend largely upon the finding of casts moulded in the renal tubules, and generally associated with albuminuria. These casts consist of a coagulable transudate, or a secretion from the diseased tubular epithelial cells, and often include or have attached to them the cells of the tubule, pus cells, erythrocytes, bacteria, granular matter, or fat originating from the degeneration of epithelial cells, etc. The casts may be short, perhaps only twice their diameter, or many times longer than the diameter. The short ones are generally straight with rounded ends, while the larger ones may be wavy or convoluted, the latter presumably from the convoluted tubules. In general the slender and faintly refracting casts are of less evil significance than the darker, granular, epithelial and fatty ones.

The best plan is to place a drop of the centrifuged sediment upon the slide without the use of a cover and examine with low power and



feeble illumination, this preliminary examination being followed by a more careful study under higher power.

A few pale hyaline casts in the centrifuged specimen are so commonly met with after a slight fever, unusual exercise or other trivial disturbance that little importance should be attached to such a finding upon a single occasion. An enormous number may be found in the urine preceding the onset of diabetic coma—even a hundred in a field under the low power, as in one of my cases. Hyaline casts are the ones most frequently seen, but the least significant in diagnosis.

**GRANULAR CASTS.**—These represent the adherence to or incorporation with the hyaline base of granular products of the disintegration of renal epithelial cells, blood cells, etc. They signify a chronic degenerative change in the kidney and are of increasing significance according to their coarser granulation and darker color. The brown granular casts are believed to owe their color to hemoglobin.

**EPITHELIAL CASTS.**—In severe nephritis of the acute or chronic parenchymatous types the cells of the tubules desquamate and adhere to the hyaline matrix and perhaps even form by themselves a hollow structure—the epithelial cast. The cells may be quite fresh, with well-preserved nuclei, or degenerated into granular material or fat. The former variety may indicate a desquamative nephritis of no extreme severity, while the fatty cast is significant of a serious lesion.

**BLOOD CASTS.**—These are seen in acute hemorrhagic processes, and are especially significant of typical acute nephritis when found with free blood cells and much albumin. They also occur in acute congestion of the kidney, hemorrhage, infarction, and possibly other conditions involving hemorrhage from the tubules. Their presence establishes the renal origin of a hematuria.

**FATTY CASTS.**—These indicate an advanced degenerative process involving the tubular epithelial cells. They are often accompanied by free fat and by free cells covered with fat droplets, and are especially characteristic of chronic parenchymatous nephritis.

**WAXY CASTS.**—These present a peculiar waxy or yellowish appearance, and may give the iodine reaction characteristic of amyloid

material. They are often of unusual size. They are of some weight in the diagnosis of waxy kidney, if associated with the usual signs of amyloid disease in the liver and spleen, but are also found in many grave cases of chronic nephritis.

**PUS CASTS.**—Pus cells escaping from a point of local suppuration into the tubules may form true pus casts, but they are not very frequently seen. A few leucocytes may be found adherent to almost any variety of casts. Shreds or clots of pus cells from pyelitis should not be confused with the true casts, which are perfectly definite in shape and appearance.

**FIBRINOUS CASTS.**—These yellowish or brownish transparent bodies are found in acute diseases of the kidney, and are much less grave in significance than the waxy casts with which they may be confused.

Castes are occasionally found made up of bacteria or of acid urates.

**CYLINDROIDS.**—These structures resemble somewhat true hyaline casts, but are longer, and taper to a point or a thread in many cases. They are probably of renal origin, but of no especial significance in diagnosis so far as we know. If in doubt we shall not err seriously if we give them the same consideration as would be accorded to light hyaline casts. The long ribbon-like shreds often found are probably of bladder origin, while the large mucous threads probably come from an irritated prostate.

**PSEUDO-CASTS.**—If a cover glass be allowed to slide over a drop of urine containing much amorphous sediment the latter may be rolled upon the slide into cylindrical masses roughly resembling tube casts. They may all point in a certain direction, and this parallelism should excite suspicion. They may completely disappear upon warming the slide. In case of doubt the physician should require for identification as tube casts that the structures examined be of regular shape, with rounded ends and parallel sides, as a rule, and, unless a fair number of casts answering this test be found, he should examine further specimens before venturing an opinion.

It has been shown by Cabot that many cases of renal disease as determined at autopsy show neither albumin nor casts during life,

and that other cases showing the latter findings proved to have no organic kidney disease. The urinary findings should always be considered in connection with the clinical features of the case, and not be regarded as of themselves of certain diagnostic importance in many or even most cases.

### THE SPUTUM

The gross appearance of the sputum should be investigated, attention being paid to quantity, color, consistence, odor, presence of "lumpy" matter, foreign bodies, etc.

The expectoration of saliva in salivation and in those chewing gum and tobacco, needs only to be mentioned. The sputum we especially consider is that coming from the bronchial tubes and the lungs. Any expectoration from the area indicated is abnormal. In young children the sputum is so constantly swallowed that it is obtained for examination only by emesis in many cases. In laryngitis, pleurisy, and early bronchitis the sputum is generally scanty. In chronic bronchitis it may amount to two pints in the day, and I have seen this amount expectorated in the so-called galloping consumption. The common limits in bronchitis, pneumonia, and phthisis lie perhaps between a half ounce (15 c.c.) and four to six ounces (120 to 180 c.c.) in the twenty-four hours. In case an empyema or liver abscess breaks into a bronchus, several pints may be expectorated in a short time, and I have seen patients drown before help could be rendered through inability to keep the air passages clear. In the acute edema of the lungs occasionally associated with mitral stenosis the pinkish frothy serum expectorated may amount to several grams per minute, as in one of my cases.

**Color.**—The sputum may be watery in appearance as in many cases of asthma; pinkish as in edema; rusty as in acute pneumonia; bloody as in hemoptysis from tuberculosis, mitral disease, or infarction of lung; "prune juice" as in low types of septic pneumonia and in gangrene of lung; of the appearance of currant jelly, as in certain cases of cancer of the lung; dark brownish, as in rupture of hepatic abscess through the lung; black from inhalation of coal dust

and powder smoke, as in coal miners; yellow from abundant pus, as in chronic bronchitis; greenish, as in gangrene, chloroma, and more often in the late stages of an influenzal bronchitis; light brown from the presence of hematin granules in the epithelial cells, as in chronic mitral disease; yellow or even greenish from bile, as in deep jaundice; bluish from an infection with the *Bacillus pyocyaneus*; and of various shades of gray, brown, and red from the inhalation and expectoration of various dusts met with in different industries by millers, iron makers, weavers, miners, stonecutters, toolmakers, etc. Malingerers may color the sputum with reddish substances to simulate hemoptysis.

**Consistence.**—The sputum in gangrene of the lung and in acute pulmonary edema may be very watery, while that of acute pneumonia may be so tenacious as to stick to the inverted cup, and all grades exist between these forms. The sputum from a phthisical cavity often appears in the form of a solid “plug,” airless and sinking in water (nummular sputum), while the yellowish purulent sputum of bronchial origin is less heavy. A mucous sputum is seen at the end of an asthmatic attack, in simple bronchitis, and in many acute infectious diseases with bronchial involvement. The watery sputum of edema may be frothy from admixture of air. The purulent sputum from liver abscess breaking into a bronchus is thicker and heavier than that from appendicular or other types of subphrenic abscess following the same course. The pus from abscess of the lung and empyema is generally so thick as to be expectorated with some difficulty. In gangrene of the lung, in putrid bronchitis, and in other processes giving rise to abundant expectoration, the sputum may separate into three layers,—an upper one of frothy mucus, a middle one of watery or muco-purulent and opaque appearance, and a bottom layer of pus, shreds of necrotic tissue, blood-clots, bacteria, etc.

**Odor and Reaction.**—Most specimens have little smell. Quite characteristic odors are those of gangrene, a sickening stench; the heavy foul odor of bronchiectasis, phthisical cavities containing decomposed secretion, abscess cavities and of putrid bronchitis; and the colon-bacillus odor of sputum from appendical abscesses and other suppurations originating in the abdomen, which have perforated into

the chest cavity. This latter contingency is probably much more frequent than is generally believed, many examples having come under my notice.

The sputum is generally alkaline excepting in those cases in which it has decomposed through long retention. Since the blood of hematemesis is generally acid from the gastric secretions, in case of doubt, as between hemoptysis and hematemesis, an alkaline reaction would point toward a pulmonary origin.

### MACROSCOPICAL CHARACTERISTICS

After spreading the sputum out upon a porcelain or glass surface, upon inspection with the naked eye or with low power lens we may learn much of value in diagnosis.

Cheesy plugs are seen in the sputum of tuberculosis, especially with cavity formation, of abscess of lung, and of gangrene. They are yellowish-gray or darker in color, and of such size as to be seen fairly with the unaided eye. They are fragments of necrotic tissue with adhering pus cells, bacteria, fatty acid crystals and epithelial cells. Dittrich's plugs may reach a larger size, and are opaque and yellowish-gray in color. They originate in the small bronchi or bronchioles, especially in severe types of chronic bronchitis, but may be expectorated free from other elements of the sputum, and even by healthy individuals. They resemble the plugs from the crypts of the tonsil. Concretions may be found coming either from the bronchi or from lung cavities. They are the result of calcification of caseous tuberculous foci in the latter, the calcareous mass reaching a bronchus by a process of ulceration. A calcified bronchial gland might easily be expectorated. Calcium salts predominate in these concretions. In one of my cases a sloughing bronchial gland was expectorated.

**Casts.**—Fibrinous bronchial casts, simple or arborescent, according to their place of origin, are occasionally seen in fibrinous types of bronchitis, and in acute pneumonia. The arborescent casts may have a definite lumen, and may branch several times, as one may note upon floating them out in water. Blood and epithelial cells are entangled or adherent. Casts of the larger bronchi may be of diph-

theritic origin, and others have been found composed of the mycelium of the *Aspergillus* and other fungi.

**Foreign Bodies.**—Pins, safety pins, fragments of nut shells, coins, pieces of bone, milk introduced into the trachea by mistake, and other foreign bodies may be expectorated and such occurrences are not infrequent. Deception by hysterical women and malingerers should be guarded against. One should be careful to distinguish food fragments and other accidental additions to the sputum from the actual contents.

### MICROSCOPICAL EXAMINATION

Epithelial and blood cells of different varieties are the most prominent objects in the average sputa. Squamous epithelia are present from the mouth, pharynx and vocal cords. Cylindrical cells are often found from the nose or the bronchial tract, occasionally showing the cilia. In bronchitis and in other forms of irritation of the respiratory tract alveolar epithelial cells are found. They often carry dark granules of carbon in those exposed to the smoke and dust of cities, giving the sputum a grayish tinge. Fat drops and myelin granules are frequently to be seen. In chronic cardiac disease we find cells containing brown amorphous pigment granules derived from hemoglobin—the so-called “heart-disease cells.” A similar staining is noted occasionally in acute pneumonia, and other processes in which blood escapes into the respiratory tract.

Leucocytes are chiefly of the polymorphonuclear and eosinophilic varieties, the former in purulent sputa, and the latter in bronchial asthma. The pus cells are often granular or fatty from degenerative processes and may contain many pneumococci, tubercle bacilli or other micro-organisms. In pulmonary tuberculosis small lymphocytes are often abundant, especially if there be no mixed infection.

Red cells are present in the sputum from any inflammatory process accompanied by rupture of minute vessels in the mucous membrane. They are often so distorted and decolorized as to be recognizable with difficulty. The greenish or yellowish-green sputum of a resolving pneumonia is stained by the hemoglobin of these cells, though the latter are not recognizable as such.

**Elastic Fibers.**—In destructive processes in the lung elastic fibers are set free and appear in the sputum, and their presence constitutes absolute evidence of the severity of the disease. In ninety per cent. of the cases tuberculous cavities are the source of origin, but they are present also in cases of pulmonary gangrene, abscess, bronchiectasis, ulceration of the larynx, etc. The wavy slender threads may have the form of the alveolar structure of the lung, indicating an advanced destruction. If the sputum be pressed between glass plates one may see the elastic tissue with the naked eye, though a low magnification is desirable. In case of doubt as to their presence one may follow the usual plan of boiling the sputum with an equal volume of ten per cent. potassium hydrate and separating the fibers by the aid of the centrifuge.

Bits of cartilage or of connective tissue may be found in the sputum from such destructive processes as give rise to elastic fibers.

Curschmann's spirals are fairly characteristic of asthmatic sputum, but may be seen in that from acute bronchitis and other conditions. They are the result of exudative bronchiolitis, and are seen under the lens to consist of a twisted fibrillary network with epithelial cells, Charcot-Leyden crystals and leucocytes, especially eosinophiles, entangled or adherent. A central thread may often be distinguished. The Charcot-Leyden crystals mentioned are seen most often in asthmatic sputum, but may also appear in that of bronchitis and tuberculosis. It is believed that they arise from the destruction of the abundant eosinophilic cells often noted in asthma.

Cholesterin plates and fatty acid crystals are seen in the sputum from cavities of phthisis and other grave destructive processes. Leucin, tyrosin, calcic oxalate and hematoidin crystals are occasionally noted.

**Animal Parasites.**—The distoma pulmonale and its ova, the echinococcic cysts, membranes and hooklets are occasionally reported. The *Entameba histolytica* is becoming more common in the examination of sputum in the United States since cases of amebic dysentery with liver abscess and rupture of the latter through a bronchus have increased in frequency, especially in those who have lived in

our tropical possessions. The eggs of the distoma hematobium are occasionally found.

**Vegetable Parasites.**—Various saprophytic organisms of the *Leptothrix* and *Streptothrix* groups, yeast fungi, sarcinae, various moulds, the *Penicillium glaucum*, and the *Oidium albicans* may be found. Certain moulds are pathogenic, especially varieties of the *Aspergillus*.

The ray fungus is more frequently detected since the recognition of actinomycosis as a not very rare pulmonary disease. The sulphur-colored granules may even be seen by the unaided eye. The frequency of actinomycosis of the jaw and the possibility of the discharge of the fungus into the mouth must be remembered.

Eisendrath and Ormsby have found the blastomycetes in the sputum of general systemic blastomycosis.

For the technique of the bacteriological examination of the sputum the reader is referred to the special works upon the subject.

**Tubercle Bacilli.**—These organisms are the most important finding in the sputum, because of the frequency of pulmonary tuberculosis and the importance of its early diagnosis. In the cheesy lumps of the sputum they are commonly abundant, but their absence does not at all exclude tuberculosis, for in many early cases of the disease none are given off into the sputum. If the examination of many slides be negative, the sputum may be boiled with sodium hydrate and centrifuged. The presence of elastic fibers in this case points toward a destructive process even in the absence of the tubercle bacilli. Care must be taken to distinguish from the tubercle bacillus the bacillus of leprosy, the smegma bacillus and the timothy bacillus.

With the tubercle bacillus in most cases one finds other organisms, especially the streptococcus, staphylococcus, pneumococcus, micrococcus catarrhalis and the influenza bacillus. Certain cases pursuing the clinical course of tuberculosis present for long periods of time only one or more of these organisms in the sputa, yet eventually the tubercle bacillus is found in nearly all. In sputum exposed to the growth of the other elements in the mixed infection, and the stain-air the tubercle bacillus is often practically smothered by the rapid



ing qualities of the tubercle bacilli are affected by the degenerative processes.

Less common than the tubercle bacillus and its associated organisms are Friedländer's bacilli, found in certain cases of lobar pneumonia; the *Bacillus pertussis*, found by Bardet and Gengou in whooping-cough; the *B. pestis* in the pneumonic variety of bubonic plague; the typhoid bacillus in cases of typhoid pneumonia and certain cases of bronchitis complicating the fever; the *B. mallei* in glanders and the *B. anthracis* in pulmonary anthrax.

## SECTION VI

### EXAMINATION OF THE SPECIAL SENSES

#### THE NOSE

THIS organ is affected either in its capacity as the seat of the sense of smell or as the beginning of the respiratory tract. The distribution of the olfactory nerve over the surfaces with which the inspired air comes in contact is an arrangement of much importance, through the protection afforded to the sensitive respiratory mucous membrane.

The connection of the nervous distribution of the nose with the vagus center and thus with the lungs through the pulmonary division of the vagus, adds an element of safety, and is at the same time of much importance because of the close connection existing between irritation of the nasal mucous membrane and hay fever and asthma.

Impairment of the sense of smell, anosmia, or loss of this sense and parosmia, or perversion of this sense, may be due to central or peripheral disease, the latter being much more frequent than the former.

Obstruction of the nares, partial or complete, is an exceedingly common occurrence. Because of its interference with the entrance of air into the lungs such obstruction in children leads to inability to nurse, and later to mouth breathing, deformity of the chest, and interference with mental and physical development. Because of the loss of the straining effect of the vibrissae on the inspired air, and of the warming and moistening effect in passing through the complicated passages of the nose, diseases of the lower respiratory tract are more frequent in mouth-breathers.

The obstruction may be from foreign bodies, as is often the case in children; from polypi, hypertrophy of the turbinate bones, deflection of the septum, rhinoliths, acute inflammation of the mucous membrane, edema, adenoids, from tumors of various kinds, and from syphilis. The snuffles of nursing babies suggests the latter cause.

The nose is the seat of many inflammations connected with the acute infectious diseases, as in coryza, measles, scarlet fever, diphtheria, and influenza. The pustules, ulcers, and yellowish discharge of glanders are rarely seen. The false membrane of diphtheria and scarlet fever, the latter also often due to the Klebs-Löffler bacillus, may be present as well. In the southwest portion of the United States, the larva of *Comptosia macellaria*, called the "screw-worm," is occasionally found, especially when the mucous membrane has been damaged by atrophic rhinitis. In tropical regions other larvæ may cause obstruction. In many of the conditions mentioned the obstructive effect of the swelling of the mucous membrane is exaggerated by the thick purulent discharge which blocks the remaining aperture.

#### EXTERNAL APPEARANCE

The shape of the nose varies greatly in different races. The most important changes in shape from the diagnostic standpoint relate to such deformities, either from turning of the nose to one side, saddle-nose, or distortion from the growth of sarcomatous or other tumors, as bring about partial or complete obstruction of one naris or even of both. Terrible deformity is occasionally seen as the result of the expansive growth of a sarcoma of the nose or other neighboring organ, and even of polypi. The tip of the nose is occasionally cut off, or one nostril slit open, in a broil or by accident; bites of the nose leave a more irregular contour than cuts or the loss from freezing. Extreme saddle-nose may be congenital, the result of syphilis, or of trauma. The congenital form shows no lesion excepting the deformity; the syphilitic nose shows the result of the loss of bone by caries, and of ulceration within the nares; the broken nose frequently occurs as the result of the kick of a horse or similar trauma, and definite healed fracture may be in evidence.

The soft tissues of the nose are thickened in myxedema and in cretinism. The organ is enlarged and deformed in rhinoscleroma. The dilated capillaries and acne of chronic alcoholism are frequently seen. If the tissues hypertrophy so that the nose becomes nodular or bulbous, the condition becomes that of rhinophyma. A slightly

cyanotic tint with dilated capillaries is to be found in many cases of mitral disease and in emphysema.

Erysipelas, furuncles, and tuberculosis, syphilis, and epithelioma present appearances similar to those observed in these affections in other portions of the body.

The alae nasi move with respiration in times of excitement, but more especially in severe dyspnea. Inspiration occasionally partially closes one nostril or even both through a valvelike action of the alae, giving at first sight the appearance of internal obstruction.

The cavities of the nose are examined by direct or reflected light, the speculum being used for the anterior nares and the rhinoscopic mirror for the posterior. The examination is much facilitated by the removal of discharges by the injection of salt solution or directly by pledgets of cotton, and by the application of a solution of cocain, with the double effect of decreasing the sensitiveness of the nose and lessening the congestion of the mucous membrane, thereby enlarging the passages. The nostril is dilated with the speculum, and the light from the head mirror reflected upon the spot to be examined. The septum, floor of the nose, and the two lower turbinate bones may now be examined. Behind the middle turbinate is the ostium maxillare, from which, in antrum disease, the discharge may be seen to escape. Discharge from the ethmoidal and frontal sinuses enters the nares above the middle turbinate and may be obstructed by a growth at this point.

Perforation of the septum is occasionally seen, generally from the gradual erosion of scab formation and removal at some point at which the mucous membrane had become damaged, with a resulting ulceration. Syphilis may also cause perforation. In the case of deflected septum "whistling" may occur as a result of a small perforation of the septum, owing to the unequal air pressure in the two nares.

Posterior rhinoscopy is much more difficult, especially because of the tendency to gag upon the depression of the tongue and the introduction of the rhinoscopic mirror. The use of cocain may be imperative. The posterior nares, middle and lower turbinates, orifices of the Eustachian tubes, polypi, fibromata, and adenoid growths may

be examined. In certain cases of syphilis and rhinoscleroma the passages may be much distorted or practically closed.

For further details as to the internal examination of the nose the reader is referred to the special works upon the subject.

### DISCHARGE FROM THE NOSE

This may be blood, the epistaxis being traumatic in origin, or proceeding from a small ulceration, generally upon the lower part of the septum. If the arteries be atheromatous the hemorrhage may become dangerous.

Bilateral epistaxis more commonly proceeds from some of the diseases giving rise to a general hemorrhagic condition, such as scurvy, hemophilia, leukemia, and purpura. In the passive congestion of mitral disease nosebleed is common. It has some diagnostic importance in the early diagnosis of typhoid, and occurs occasionally in many other acute infectious diseases. The epistaxis of the full-blooded, that from exertion, especially in mountain climbing, and the vicarious form in suppression of the menses, should be mentioned. The possibility of the escape of blood into the pharynx, whence it may pass to the stomach and be subsequently vomited, is of importance in the diagnosis of the causes of hematemesis.

Purulent discharge may be found in acute and chronic rhinitis, as a result of irritation by foreign bodies (unilateral) or of the discharge of sinus disease, either maxillary, frontal, or ethmoidal (generally unilateral). Irritation of the skin about the orifice, with cracks and fissures, may be noted. Offensive discharge is present from both nostrils in atrophic rhinitis, in nasal diphtheria, glanders and in cases of syphilis and malignant growth. If caries of bone occur, the odor may become extremely fetid:

Watery discharge is common in coryza, iodism, hay-fever, and in infectious diseases accompanied with nasal congestion. In certain rare cases cerebrospinal fluid escapes from the nose through the cribriform plate of the ethmoid bone. One should not assume watery or other discharge to be of neuralgic origin without being careful to exclude local disease of the nose or accessory cavities.

Fluids are regurgitated through the nares when the palate is

cleft, and when it fails to fulfil its function, from diphtheritic or bulbar paralysis.

### SENSE OF SMELL

The sense of smell is tested by the inhalation (sniffing) of odoriferous substances which are not sufficiently pungent to irritate the distribution of the trigeminal nerve. The volatile oils serve the purpose well. Extreme acuity of the sense of smell is sometimes seen in neurotic and hysterical patients (hyperosmia). The inhalation of the strong and especially pungent odors causes temporary suspension of the function of smell from over stimulation, but it returns in a few minutes in most cases.

A common hallucination in mental disease is that of smelling odors which have no real existence (kakosmia). The sensation of perceiving a disagreeable odor may constitute the aura of epilepsy.

### THE EYE

Entirely apart from the disturbances of vision which occupy the attention of the ophthalmologist, there are many symptoms relating to the eye of the utmost importance to the clinician, especially in the investigation of the nervous and vascular systems. The common manifestations of disease as presented in the examination of the eyelids have been noted, in the chapter upon "Examination of the Patient," page 13. Edema from renal and cardiac disease and from anemia, the swelling of angioneurotic edema, the puffiness of arsenical poisoning, that of whooping cough, with possible ecchymosis, edema from exposure to electric light, from eye-strain in refractive errors, from antral disease, from coryza, iodism, measles, trichiniasis, and from inflammation of the tear ducts, should be recalled. The latter is occasionally mistaken for facial erysipelas. The unique and repulsive edema about the eye and nose, seen in thrombosis of the cavernous sinus, and usually associated with exophthalmos, is of great importance in the diagnosis of this trouble. Thrombosis of other cerebral sinuses may give rise to a somewhat similar appearance, the internal nasal and other veins furnishing means of anastomosis between the superficial veins of the face and the sinuses.

### THE EYELID

The margin of the lids is often chronically inflamed (blepharitis trachoma) and frequently misplaced lashes irritate the corneal surface. Upon the margin of the lid we may see a sty (hordeolum) or abscess of the lid, or the inflamed and cystic Meibomian glands, chalazion. Warts and epitheliomata are not uncommon, and the latter may develop from the former. The initial lesion of syphilis is occasionally seen. Tertiary ulceration and various acute infections may be noted. Inflammations and ulcerations are likely to evert the lids, owing to cicatricial contraction.

Inflammation of the tarsus may be due to syphilis or tuberculosis, and topi may be found here in gout. Herpes zoster ophthalmicus resembles the herpetic eruption elsewhere, and is generally accompanied by much pain and adenitis. The first and second divisions of the fifth nerve are involved. Corneal ulceration and iritis may be serious complications.

The soft and loose tissues about the eye become the seat of extensive extravasation of blood upon comparatively slight trauma. The occurrence of surgical emphysema signifies a fracture of the wall of the orbit, air entering the tissues upon blowing the nose.

Upon both upper and lower lids toward the inner angles we often see the buckskinlike patches of xanthoma or xanthelasma. They occur especially in women of sluggish digestive habit, and should suggest the possibility of gall-stone disease. The pediculus pubis and its eggs may be found upon the eyelashes.

Sebaceous cysts are not infrequent. Chronic inflammation of the tear gland is present in Mikulicz's disease, and either acute or chronic inflammation of the gland or its duct may occur in other connections. The obstruction of the tear duct, with epiphora, leads to excoriation of the skin from the irritation of the overflowing tears.

Darkening about the lids and under the eyes is especially common in women with pelvic disease and chronic constipation, or other digestive disorders.

Ptosis, or drooping of the upper lid may be congenital, but it is commonly a part of oculo-motor paralysis, and suggests an inquiry

as to the presence of syphilis. The congenital cases are due to defect or absence of the levator palpebrae muscles. Bilateral ptosis may be associated with other manifestations of hysteria. It may be present as a transient symptom, especially on awakening, in feeble individuals, and after the use of gelsemium and conium. It often occurs in bulbar paralysis and myasthenia gravis.

The term lagophthalmus indicates an inability to close the upper lid, and is one of the manifestations of facial paralysis. In protrusion of the eyeball, it is of purely mechanical origin and constitutes Von Graefe's sign in exophthalmic goiter, the lid being unable to follow the downward movement of the eye. In exhausting disease, as in tuberculosis in adults, and in many serious conditions in children, the lids often remain partly open during sleep, exposing the white sclera.

Blepharospasm, or spastic closure of the eyelids, is common in acute inflammatory conditions of the eye, and in the over sensitivity to light called photophobia, so often seen in meningitis and general diseases causing meningeal irritation. It is often a "habit spasm," and may be present in migraine and hysteria.

Reflex irritation of the fifth nerve may cause obstinate spasm of the orbicularis muscle.

### THE EYE-BALL

**Exophthalmos.**—Protrusion of the eye-ball results from increased pressure behind the eye, or from paralysis of the ocular muscles which normally hold it in place to some extent. The increased pressure may be from the most various causes. Tumors of bony or soft parts, hemorrhage, aneurism, cellulitis, or suppuration in the orbit or extending into it from surrounding sinuses may cause it, these factors acting more especially to cause unilateral proptosis. Bilateral protrusion is seen most commonly in exophthalmic goiter, and occasionally in those suffering from dyspnea, as in asthma, in scurvy, from hemorrhage under the orbital periosteum, in chronic nephritis, and in cardiac disease. The protrusion is accurately measured by the ophthalmologist, so that one need not be deceived by the undue exposure of the sclera by retraction of the lids.



**Enophthalmos.**—Enophthalmos or recession of the eye-ball is a much less important symptom. It is bilateral when arising from lessening of the orbital fat, as in phthisis and other wasting diseases, or from lessening of the body fluids, as in cholera and hemorrhage. Unilateral recession may be noted in facial hemiatrophy, in disease of the sympathetic and after trauma.

### CONJUNCTIVA, SCLEROTICA, AND CORNEA

**The Conjunctiva.**—The conjunctiva is reddened in those conditions in which the vessels are dilated, as in disease of the cervical sympathetic, and in alcoholics, but especially in those in whom this dilatation comes from an active inflammation, as in simple conjunctivitis or the form which accompanies coryza, measles, yellow fever, meningitis, and typhus fever. The Koch-Weeks bacillus, the pneumococcus, the gonococcus, and other organisms produce specific forms of conjunctivitis of more especial interest to the ophthalmologist. Swelling of the lids with mucopurulent discharge accompanying the inflammation, reaches its acme in ophthalmia neonatorum and in the gonorrheal ophthalmia of adults. Exposure of the eye from facial paralysis, damage from foreign bodies, congestion about an ulcer, and the reddening in glaucoma, should be noted. Diphtheritic infection occurs upon this mucous membrane but rarely.

Hemorrhages frequently occur from local trauma, and in conditions raising the general blood pressure, notably in whooping-cough, vomiting, and severe muscular strain. Hemorrhagic infarcts occur in septic endocarditis. In fracture of the anterior part of the base of the skull, blood may creep forward under the conjunctiva, constituting an important diagnostic feature. When the vessels become weakened in advanced arteriosclerosis, spontaneous hemorrhage may be seen.

The eye exposed by failure of the normal function of the lid becomes inflamed, and often dryer than normal. The glazed eye of collapse and the moribund state is of importance. The moistening of the eye in emotion is physiological.

Lacrimation arises from irritation of the conjunctiva, either local

in character or as a part of general disease. The conjunctiva and sclera may be yellow in jaundice, this sign being of especial value in other than white races. The bluish-white color of anemia and certain wasting diseases is of little import. A gouty deposit is occasionally seen in the conjunctiva. Pterygium and other growths are of more especial interest to the specialist.

**The Cornea.**—The medical diagnostician should inspect the cornea for ulcers, keratitis, and the arteriosclerotic change designated “arcus senilis.” The exposed cornea in exophthalmic goiter and typhoid and after paralysis of the seventh nerve, the anesthetic one in paralysis of the first division of the fifth nerve, or after removal of its ganglion for neuralgia, and in hysteria, the inflamed cornea after trauma and from the effect of the eruption of small-pox, and the cornea of lessened resisting power, as in tuberculous children, are the especial seats of ulceration. The dendritic ulcer is the result of trophic disturbance in the distribution of the fifth nerve.

Keratitis of the interstitial type is of interest as indicating a probable hereditary syphilitic infection. It is usually bilateral, occurs in children, and is commonly associated with other signs of a luetic heredity. The vascularized ground-glass opacity of the cornea, less opaque in certain areas, is characteristic.

**ARCUS SENILIS.**—A congenital form exists. Much dispute occurs as to the significance of the acquired grayish ring, at times incomplete, around the cornea, frequently present in senility. There can be no question that it is a degenerative change, whether or not it is always due to arteriosclerosis, and the subject of it is not a fit candidate for life insurance in my opinion, whether appreciable hardening of the arteries is found or not. I have seen it as early as the fourth decade, especially after chronic exhaustive diseases. The false arcus, due to fatty deposit, and more yellow in color, should not be mistaken for the true form.

## THE IRIS

Irides of different color are not uncommon, and are reckoned amongst the stigmata of degeneration. Deformity as the result of iridectomy or trauma, and adhesions and consequent irregularity

in the shape of the pupil, should be sought for. Iritis is sufficiently common in syphilis and certain toxemias to lead the clinician to investigate such sources of origin before referring the cases to a specialist. The chronic rheumatic affections, tuberculosis, and gout should also be considered.

### THE PUPIL

We should examine the pupils for evidence of dilatation or contraction, inequality (anisocoria), irregularity of margin, response to light and accommodation, hippus, and for the skin reflex. The parallelism or otherwise of the eyes is commonly noted during this examination.

**Dilatation.**—Dilated pupils are seen most commonly as the result of the use of mydriatics for examination of vision, or of cocain (often unilateral) for the removal of a foreign body. Cocain habitués show mydriasis when under the influence of the drug. In neurotic and hysterical individuals, in many tuberculous subjects, and in children, dilated pupils are common. In anesthesia from chloroform and ether, sudden dilatation of the pupils is an ominous sign.

In optic atrophy and in blindness from other causes the pupils dilate, and the light reflex fails. In cerebral anemia from any cause, in severe dyspnea, and as the result of gross disease in the brain or meninges, dilatation is often seen, due either to irritation of the dilating mechanism of the pupil or to paralysis of the contractor fibers.

In certain types of insanity, after epileptic convulsions, and in diphtheritic paralysis, dilatation of both pupils is frequent. Unilateral dilatation is seen in paralysis of the third nerve, after trauma of the eye-ball, in cataract, optic atrophy, and glaucoma. Irritation of the cervical sympathetic by aneurism or other cause gives dilatation upon the affected side.

If one optic nerve be destroyed the dilated pupil upon that side fails to contract upon the stimulation of light, but responds consensually upon the stimulation of the opposite eye, the oculomotor nerve and its connections being intact.

**Contraction.**—The pupil contracts from paralysis of the sympa-

thetic upon that side, as is often seen in aneurism, or from irritation from the center controlling the contractile fibres of the iris, as in many irritative brain and meningeal diseases. The pupils are smaller in age than in youth. Spinal myosis is frequent in tabes and other cord diseases. Extreme contraction is seen in poisoning by opium, eserine and pilocarpin. Enlargement of the vessels of the iris, as in iritis, and conditions of venous congestion, produce myosis. In hemorrhage into the pons, uremia and sunstroke, contracted pupils are often of value in diagnosis.

**Anisocoria.**—Anisocoria is not necessarily pathologic, and is even quite frequent in healthy individuals at high altitudes. Any of the causes of dilatation or contraction of the pupil, if acting unilaterally, may bring about inequality of the pupils. I have seen aortic aneurism cause dilatation of one pupil through stimulation of the sympathetic and contraction of the other through paralysis from pressure, at the same time.

Unequal pupils in the absence of local eye disease should suggest to the internist the possibility of meningeal or cerebral disease, especially unilateral in character, aneurism, and the late syphilitic diseases, tabes and general paralysis. In pneumonia and pulmonary tuberculosis the anisocoria is of little practical importance. It is present occasionally in exophthalmic goiter.

The pupils should receive equal illumination before deciding that they are unequal in size.

**Irregularity of Margin.**—Irregularity of the margin of the pupil is most frequent as the result of adhesions from a previous iritis. Because of the occurrence of this irregularity in syphilitics we should always think of the possibility of tabes, tabetic abdominal crises, general paralysis or other late syphilitic disease, when it is observed. Thus I have seen many cases in which irregularity of the pupil margin with anisocoria was sufficient to lead to the detection of the tabetic gastric crises in patients wrongly treated for gastric ulcer or gall-stones.

**Response to Light and Accommodation.**—The former is tested by allowing light, as by withdrawing the hand from over it, to fall suddenly upon the open eye. The pupil contracts in health—the

direct light reflex. The eye still covered should contract, although not directly illuminated—the indirect reflex.

Response to accommodation may be tested by asking the patient to look suddenly at the finger tip held a few inches away, after he has relaxed his ciliary muscle in looking at a distant point, as some object seen out of the window. The pupil should contract visibly with the effort at accommodation.

**Skin Reflex.**—The sensory reaction follows stimulation of the skin of the neck, by pinching or otherwise, causing irritation of the cervical sympathetic, and dilatation of the pupils.

The size of the pupil depends upon the balance between the dilator or radial muscular fibres of the iris, and the contracting or circular ones, the former governed by the sympathetic, the latter by the third nerve. Thus paralysis of the third nerve may give rise to dilatation by permitting the sympathetic to act unopposed, while paralysis of the sympathetic may lead to contraction, through unopposed action of the oculomotor mechanism. Stimulation of the oculomotor center causes a direct contraction, as stimulation of the sympathetic causes a direct dilatation; the opposing force in each instance being overcome. The iris may fail to respond to any stimulus because of the adhesions of an old iritis, or other local disease.

**Pathological Reactions.**—**HIPPU.**—Rapidly varying dilation and contraction of the pupil without change of illumination is often seen in nervous individuals and in children, without pathological significance. It is an interesting but not particularly valuable phenomenon in epileptics, hysterics and neurotic patients in general, and is frequently seen in pulmonary tuberculosis, in mania and in meningitis.

**ARGYLL-ROBERTSON PUPILS.**—We have in this case loss of reaction to light with preservation of the reaction to accommodation and convergence. It is an almost constant phenomenon in tabes and is seen frequently in parietic dementia. Contraction of the pupils and frequently irregularity of the margin of the pupil may be present. The conditions in which this reflex immobile pupil are found are so often syphilitic in nature that we are justified in as-

suming a luetic lesion unless the most positive evidence to the contrary is presented.

The opposite condition—preservation of the light reflex with accommodative iridoplegia is of vastly less value in diagnosis, and much less common. It may be seen in certain diseases of the oculomotor nucleus and in post-diphtheritic cycloplegia.

### OCULAR MOVEMENTS

Under normal conditions the eyes move in perfect accord, through the action of a complex and delicate mechanism. So many different parts of this mechanism may be disarranged in disease, with perfectly characteristic symptoms as a result, that much assistance in diagnosis may be available through a careful study of the anomalies in the ocular movements. The coördinative apparatus having for its object the focusing of an exact image upon the fovea centralis of each eye is described in the special works upon the subject.

**Strabismus.**—The squint may be convergent, divergent, or vertical, in the latter case either upward or downward. The sound eye is exactly focused upon a given object, while the other wanders from it, although in functional strabismus the squint may be alternating, either eye being fixed since they are in this case of approximately equal acuity.

Concomitant or functional strabismus is seen when the two eyes are so unequal in power that no advantage is gained by the attempt to form a single image; when errors of refraction disturb the normal relation between convergence and accommodation, and when opposing muscles fail for any reason to act together. In concomitant strabismus, a convergent type seen in young children, as distinguished from the paralytic form, the affected eye moves with the normal one, but not in exact focus, and the deviation is approximately the same whichever eye is tested. The absence of double vision is the most striking clinical feature in the differentiation of this type of strabismus from the paralytic form.

Paralytic strabismus is characterized by the more or less complete loss of power in one or more of the external ocular muscles, so that certain movements of the affected eye are impossible, and dip-

lopia results when these movements are attempted. This may be a purely functional and temporary phenomenon, as in alcoholic intoxication, and after the use of gelsemium, conium and belladonna.

**Ocular Paralysis.**—**DIPLOPIA.**—Diplopia is the most important symptom of paralysis of one or more of the external ocular muscles, since such disability prevents that formation of the exact image upon the fovea of each eye essential to perfect binocular vision. In connection with diplopia we also have the inability of the diseased eye to make certain movements, paralytic strabismus, false projection of external objects and, because of the fact that the balancing mechanism of the body is dependent upon correct perceptions of the relations of surrounding objects, ocular vertigo.

Vertigo may be present only when vision is attempted with the diseased eye alone. The patient may place the head in an unusual position in order to use the ocular muscles to the best advantage, and thus lessen the diplopia and vertigo.

In paralysis of one external rectus homonymous diplopia occurs, and crossed diplopia if an internal rectus be involved. Vertical diplopia of different types depends upon paralysis of one superior rectus or one inferior oblique muscle.

**THIRD NERVE.**—Paralysis of the third nerve is characterized by ptosis, external strabismus, diplopia, dilated pupil, reacting neither to light nor accommodation, and oftentimes slight proptosis. The paralysis is often more or less incomplete. It may be peripheral, presumably rheumatic, and following exposure to cold, but is much more often due to syphilitic disease involving the origin of the nerve. Functional temporary paralysis occurs rarely in migraine; and may recur periodically. If associated paralysis of the fourth and sixth nerves be present, the trouble is probably definitely organic, and due to disease affecting the nearly related nuclei of these three nerves.

In unilateral disease of the crus the third nerve may be paralyzed upon that side with hemiplegia of the opposite side. Oculomotor palsy may be seen in tabes, progressive muscular atrophy and bulbar paralysis. Diphtheria, diabetes, meat poisoning and other conditions such as may give rise to multiple neuritis, occasionally affect this nerve.

**FOURTH NERVE.**—The paralysis of the superior oblique muscle, which this nerve supplies, causes double vision upon looking downward, the image of the affected eye being the lower one. This nerve is so well protected within the brain that isolated paralysis is comparatively rare. Any of the causes of third nerve palsy may affect it. Pressure may occur upon this nerve alone in case of tumor of the under surface of the anterior lobe of the cerebellum, and it may be destroyed in fracture of the skull.

**SIXTH NERVE.**—Paralysis of this nerve is the most common of the ocular palsies, owing to its long and exposed course at the base of the brain. Fracture of the base, hemorrhage, meningeal exudate and gumma are frequent causes of paralysis. The external rectus being paralyzed, internal strabismus results, with diplopia. Lesions producing unilateral damage to the pons, and giving rise to crossed paralysis, may present abducens paralysis upon the side of the facial palsy from the involvement of the nerve at that point. Various toxemic conditions may affect the sixth nerve or its nucleus. Diabetes mellitus may cause a temporary paralysis, often accompanied with pain. I have seen the nerve paralyzed in anterior poliomyelitis. If the third, fourth and sixth be involved we have total ophthalmoplegia externa. Internal ophthalmoplegia designates paralysis of the iris and ciliary muscle. A basal lesion may involve the facial nerve with those supplying the ocular muscles, producing facial and ocular paralysis without hemiplegia. Either variety of ophthalmoplegia may be a part of an upper bulbar palsy. The assistance of the specialist is indispensable.

**Conjugate Deviation.**—The internal rectus of one eye and the external rectus of the other are so associated in their action that spasm or paralysis of such a pair of muscles causes conjugate deviation, the spasm overcoming the opposing muscles, while the paralysis gives the full control to the unparalyzed pair. In irritation of the cortical controlling center, as in epilepsy, the eyes turn away from the lesion, while destruction of the cortical center or of the path of communication with the ocular muscles, as in apoplexy, causes the patient to "look toward the lesion." The nature and location of the process causing conjugate deviation may generally be determined



by associated symptoms which indicate apoplexy, tumor, meningitis, etc.

**Nystagmus.**—This involuntary oscillation of the eye-balls is more commonly lateral than vertical or rotary. It is due to bilateral spasm of the ocular muscles, of central origin, and may be either functional or organic. It is a common symptom in blindness. Coal miners, from over strain of the eye muscles in the awkward positions in which much of their work is done, develop it. It is seen in hysterical, neurotic, hydrocephalic and insane persons, but is of clinical value chiefly in disseminated sclerosis and Friedreich's ataxia. It is occasionally seen in connection with basal meningitis and with tumors involving the crura cerebri, pons and cerebellum.

**Muscular Asthenopia.**—Muscular asthenopia is of great importance to the internist, since it may cause ocular headaches, nausea, vertigo, insomnia and even neurasthenia. Its investigation should be undertaken by a trained ophthalmologist, since errors of refraction and other special features must be carefully considered.

## VISION

The imperfections of vision due to refractive errors, cataract, opacities of the cornea and vitreous humor do not especially concern us here, excepting as they induce headache and other nervous disorders through the strain attendant upon futile efforts to obtain clear vision. Xanthopsia from the use of *santonin* or from jaundice; *muscæ volitantes*, the *scotomata* preceding migrainous attacks, and various flashes of light and other functional disorders associated with different nervous diseases, heart diseases, nephritis, indigestion, and cerebral anemia, may be mentioned, but are not of great importance.

**The Fields of Vision.**—HEMIANOPSIA.—This term designates blindness in one half the visual field, being right or left sided as the patient sees, not as the retina actually receives impressions. It is due to functional or organic disease affecting the apparatus of vision between the chiasm and the occipital lobe.

The most common and important variety is the homonymous form, in which either the *right* or *left* sides of both retinae are in-

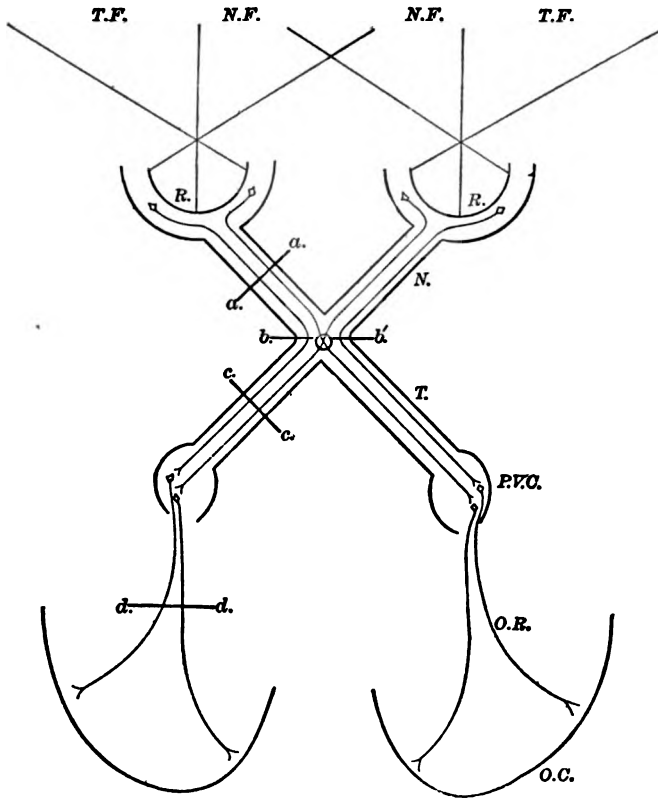


FIG. 6.—DIAGRAM OF LESIONS PRODUCING HEMIANOPSIA. *T.F.*, temporal field; *N.F.*, nasal field; *R.*, retina; *N.*, optic nerve; *T.*, optic tract; *P.V.C.*, primary visual centers; *O.R.*, optic radiations; *O.C.*, occipital cortex; *a.a.*, lesion producing unilateral blindness; *b.*, lesion producing unilateral hemianopsia; *b.b'*, lesions producing binasal hemianopsia, very rare; *O.*, at center of chiasm, lesion producing bitemporal hemianopsia; *c.c.*, *d.d.*, lesions producing homonymous hemianopsia. Hemianopic pupillary reflex in lesions anterior to primary visual centers, and behind the chiasm. (Osler: "Modern Medicine," vii.)

involved, producing respectively *left* or *right* lateral hemianopsia. In bi-temporal hemianopsia the outer half of each field of vision is affected, and the contrary condition holds in bi-nasal hemianopsia. The superior and inferior varieties of hemianopsia are rarely seen.

The location and extent of the loss of vision are roughly tested in each eye separately, after covering the other, by the common method of bringing a bit of card or the finger toward the center until

detected by the patient, the latter's eye being meanwhile steadily fixed upon the physician's head or face.

Functional hemianopsia may be due to hysteria, migraine or gout; most cases are of organic origin, and due to gross disease, as tumor, hemorrhage, softening, meningeal exudate, etc. The nature of the cause may be inferred from associated symptoms, but is often uncertain.

The lesion causing homonymous hemianopsia affects the opposite optic tract or the fibres passing through it at some point posterior to the chiasm, since all the fibres at this point go either to the right sides or the left sides of the retinae, as the case may be.

Binasal hemianopsia is caused by symmetrical lesions at each outer side of the chiasm or the outer sides of the optic nerves. Because of the rarity of such lesions this type of hemianopsia is infrequent.

Bitemporal hemianopsia is caused by a lesion of the crossed fibres only at the chiasm, the function of the nasal halves of the retinae being thereby suspended. The destruction may be due to tumor, the enlargement of the pituitary body in acromegaly, aneurism, fracture or vascular disease.

In relative hemianopsia recognition of color only is lost, perception of light and form being preserved.

If the lesion be in front of the primary optic centers, the pupil reacts when a beam of light is thrown upon the sound side of the retina, but not in illumination of the blind side (Wernicke's hemianopic pupillary inaction). This delicate test is better left to the specialist.

In those cases of hemianopsia due to organic brain disease in the internal capsule, hemiplegia and hemianesthesia, with aphasia in left-sided lesions, are present. In the absence of motor and sensory symptoms we should look for the lesion in or near the cuneus. Incomplete hemianopsia with mind blindness or word blindness signifies that the lesion is cortical.

If the entire chiasm be destroyed or bilateral lesions of the cuneus be present, total blindness results. Unilateral blindness may be due to disease of one occipital lobe or one optic nerve.

**Amblyopia and Amaurosis.**—Amblyopia signifies partial blindness, and amaurosis complete blindness, without organic lesions determinable by the ophthalmoscope. Such defective vision is due to damage from malnutrition of the ganglionic cells of the retina, after severe hemorrhage or from spasm of the retinal vessels; to the toxic action of methyl alcohol, lead, quinin, tobacco and the salicylates; to the poisons of diabetes and uremia; to trauma and electric shock; to retrobulbar optic neuritis, either acute or chronic, and sometimes hereditary; but it is most often seen in connection with hysteria and migraine. Color blindness is a partial congenital amblyopia, as is the imperfect perception of form due to strabismus or high refractive error.

Amblyopia and amaurosis are often sudden and temporary since many of the causes mentioned are subject to great variation, or to recovery.

Contractions of the visual field for light and for color are determined by the specialist by means of the perimeter. They are especially seen in hysteria and in the traumatic neuroses. The toxic amblyopias show central scotomata for certain colors.

Sudden or rapid loss of sight should lead to examination for retinal hemorrhage, embolism or thrombosis of the arteria centralis retinae or its branches, uremia or other toxemic process, poisons from without, notably wood alcohol and quinin, and the functional neuroses.

The frequent association of diabetes and cataract should be noted. Glaucoma is frequently mistaken for neuralgia or some other painful affection, and therefore the occurrence of severe pain, congested conjunctiva, blurred vision, dilated pupil, and increased intra-ocular tension with nausea and vomiting should call for careful examination of the eye by a competent specialist.

### OPHTHALMOSCOPIC EXAMINATION

Certain retinal appearances are of great value to the internist in the diagnosis of particular diseases. A reasonable familiarity with the use of the ophthalmoscope should enable him to recognize the conditions to be mentioned, although I have made it a rule to refer

all cases of importance to an ophthalmologist for more expert opinion.

**Retinal Hemorrhage.**—Red hemorrhagic patches, generally irregular in outline, are common in heart disease, severe anemias and other blood diseases, and in chronic nephritis, especially with degenerated arteries and high blood pressure. Malignant endocarditis and pyemia lead to embolism, with attendant localized blood stasis in the retina.

Pulsating retinal arteries are to be seen in aortic regurgitation, and occasionally in neurasthenia and exophthalmic goiter. The rosy optic disc of neurasthenia should be mentioned.

**Optic Neuritis.**—Inflammation of the retrobulbar portion of the nerve occurs in tobacco and lead poisoning, syphilis, gout, diabetes and other diseases. Papillitis, or inflammation of the nerve head, presents a marked swelling, the so-called "choked disc." During the active stage the disc is swollen and reddened and the margin is blurred. With the proliferation of connective tissue which occurs with the subsidence of the inflammation, the nerve head shrinks and becomes pale—a secondary optic atrophy. In tabes, paresis and disseminated sclerosis, after hemorrhages and in certain toxemias a primary atrophy occurs.

The choked disc is especially significant as a sign of brain tumor, cerebral abscess or meningitis.

Retinitis and neuroretinitis are generally of anemic, nephritic, leukemic, syphilitic or toxemic origin, and in many cases are easily recognized. The occurrence of miliary tubercles in the choroid, obstruction of the retinal vessels, and more especially arteriosclerotic changes, may be of much importance to the clinician, but the assistance of the specialist should generally be obtained.

## THE EAR

The appearances of the external ear are discussed elsewhere. Unusually prominent, misplaced or imperfectly developed ears are often found in those mentally or morally defective.

The size and shape of the canal should be noted, and whether

eczema, cerumen, dirt, foreign body, abscess, furuncle, or serous, purulent or bloody discharge be present. Practice will enable one to manipulate the conical speculum and the auricle with one hand, leaving the other free for further procedure.

Serous discharge is often seen in fracture of the base of the skull, and hemorrhage occurs in the same cases. Slight hemorrhage may be noted in hemophilia and after traumatic rupture of the drum. Pus occurs most often from abscess or furuncle of the meatus, or otitis media, but may come from a parotid or other abscess here. The very great frequency of otitis media in children even with no especial indications excepting fretfulness and fever should lead us to a very careful examination of the ear in case of doubt. If no pus be found deep in the ear the reddened and bulging drum membrane may be seen if a good light be focused upon it.

### DEFECTS IN HEARING

These are due either to the defects in the sound-conducting apparatus or in the nervous mechanism, including the auditory nerve, its nucleus and its cortical center, the latter form being termed nervous deafness. In this type the vibrations of the tuning fork or ticking of a watch are not heard even upon bony contact, since the perceptive apparatus is defective, while in middle-ear deafness the contact with the mastoid enables bone conduction to replace the normal aerial conduction.

Most cases of deafness are of the middle-ear type, and due to obstruction in the canal or to inflammatory conditions in the middle ear, the latter being especially common after the eruptive diseases of childhood, in tonsillitis, rhinitis, adenoid disease, and nasal obstruction from any cause.

Nervous deafness in a majority of cases is of labyrinthine origin, the terminations of the auditory nerve in the internal ear being at fault. They may have been damaged by exposure to too severe shock, as in boilermakers; hyperemia of the labyrinth, as the result of drug action, notably from quinin and the salicylates may be present; or the filaments may have suffered from tuberculosis or syphilitic disease, or become atrophied in locomotor ataxia. Mén-

ière's disease and some of the low fevers should be mentioned under this heading. Lesions of the nucleus and root of the nerve occur from trauma, pressure by tumor or otherwise, and in meningitis. Deaf-mutism is often the result of the damage produced in infants by epidemic meningitis.

Disease of the left cortical center produces word deafness, and hysterical deafness has the same point of origin. Various coarse organic brain lesions may by pressure produce cortical deafness by destruction of the fibers running to the nucleus of the nerve, as is notably the case in brain tumor.

Hyperacusis may be present in hysteria, meningitis and facial paralysis. Dysacusis, in which ordinary sounds produce unpleasant auditory sensations, is common in migraine, hysteria, neurasthenia, and various conditions involving nervous irritability.

### TINNITUS

Subjective sounds of widely different character heard by the patient in one or both ears, or in the head, are called tinnitus. They may arise from many different causes. These commonly affect the circulatory condition within the ear, as after the use of drugs, in arteriosclerosis and high arterial tension, or are associated with increased irritability of the nervous system, as in hysteria, neurasthenia, migraine, etc., or are toxic, as in gout, alcoholism and tobacco poisoning. In Ménière's disease tinnitus is often overshadowed by vertigo and deafness.

This distressing symptom commonly demands the services of a specialist, and the same remark applies to most of the internal auditory conditions mentioned.

### THE LARYNX

The examination is made with the usual laryngoscopic mirror illuminated by a good light reflected from the head mirror worn by the operator. The tongue is better drawn forward by the fingers with the aid of a napkin. The throat mirror is then passed over the base of the tongue avoiding contact with any of the parts until

it raises the soft palate and uvula and brings into view the epiglottis and vocal cords. The condition of the dorsum of the tongue and the lingual tonsil, and any ulcerations or varicose veins should be noted. In the depressions of either side are often found fish bones and other foreign bodies which may now be detected. The epiglottis comes most prominently into view, and infiltration and ulceration are to be especially noted. The aryepiglottic folds and pyriform sinuses are seen laterally from the glottis.

The vocal cords attract attention from their white appearance, contrasting sharply with the surrounding parts. They reach from their forward attachment to the thyroid cartilage to the arytenoid cartilages behind. They should in health be centrally placed and moved symmetrically, approaching to central contact during phonation. The anterior attachment may be concealed by the epiglottis. If the patient be now instructed to say "E" the position of the epiglottis changes so that the examiner may see the previously covered portion of the cords and the under surface of the epiglottis. The mobility of the vocal cords and their general relations to the surrounding parts are best tested by instructing the patient to say "Ah."

During quiet respiration the separation of the cords at their posterior attachment permits of a view into the trachea to the bifurcation under favorable conditions, the lateral ventricles and the false vocal cords appearing laterally. The use of the autoscope and the bronchoscope, from which great help may be obtained in certain cases, especially in localizing and removing foreign bodies, is necessarily confined to operators of especial skill. One should note the color of the cords, becoming reddish in acute laryngitis, the presence of tumors, of nodules on the free edge, and the mobility of the cords in phonation and respiration.

Tuberculous, syphilitic or malignant ulceration may be noted on the epiglottis. Congestion and edema may be present from circulatory disturbances, from acute infectious diseases, especially diphtheria, and from irritation of steam, acid vapors or other traumatism. The swelling in connection with angioneurotic edema may be fatal.



### LARYNGEAL PARALYSIS

The recurrent laryngeal nerve supplies all the muscles of the larynx except the cricothyroid, supplied by the superior laryngeal. The spinal accessory nerve furnishes the motor filaments to those branches of the vagus. Paralysis of the larynx may thus originate in:

(1) Lesions affecting the nuclei in the medulla, as in bulbar paralysis.

(2) Through pressure upon the vagus or spinal accessory nerves in their course, as in tumors of the neck, or in those operative cases, especially involving the thyroid gland, in which a ligature includes by mistake the vagus nerve, or it is otherwise damaged.

(3) Through pressure upon the laryngeal branches, especially the inferior, because of its long and exposed course, from aneurism, enlarged bronchial glands, goiter or tumor of any kind, contracting pleural adhesions, large pericardial effusion, or great dilatation of the left auricle. The left nerve, passing below the aortic arch, is especially exposed in the intrathoracic conditions mentioned, while the right may be involved in subclavian aneurism.

(4) The paralysis may be toxemic as in diphtheria, alcoholism and lead poisoning.

(5) Hysterical aphonia and that due to inflammation of the structures of the larynx should be mentioned.

Paralysis of the recurrent nerve upon one side causes the corresponding cord to remain motionless in the cadaveric position, as is especially seen upon the left side in aneurism of the aorta; the double form is oftener the result of diphtheritic neuritis or central disease than of local pressure. In the unilateral form the unparalyzed cord passes beyond the median line to meet its fellow, and thus restores phonation in many cases, though not perfectly. In the double form, aphonia is complete, and the cords can neither be brought together for effective cough, nor separated to allow deeper breathing upon severe exertion.

Paralysis of the abductor or posterior crico-arytenoid muscle leaves the affected cord in the central position, and thus interferes

with inspiration. This interference threatens suffocation if the paralysis be bilateral. Expiration, coughing, and phonation are not especially compromised. The unilateral form is especially significant of pressure upon one recurrent nerve, the left being most frequently involved. The double form is generally central, and may be hysterical.

Paralysis of the lateral crico-arytenoid and the associated muscles prevents the cords from attaining the central position in phonation—adductor paralysis. This is commonly bilateral, and is the especial form seen in hysteria. Phonation only is affected, as may be noted by observation of the normal movement of the cords during respiration. This type of paralysis is seen also in acute laryngitis and after abuse of the voice. If the internal thyro-arytenoids alone be involved, the approximated cords leave an elliptical opening at the center; if the interior arytenoid muscle is paralyzed a triangular opening is left posteriorly.

Gowers' table gives an excellent summary of the conditions discussed:

SYMPTOMS	SIGNS	LESION
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line, in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy.

SYMPTOMS	SIGNS	LESION
No voice; perfect cough; no stridor or dyspnea.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

### VOICE

Hoarseness arises when the function of the larynx is so interfered with that dysphonia results, the cause being either local laryngeal disease or laryngeal paralysis. If the interference be sufficient to prevent speech we have aphonia.

Laryngitis of some type is the most common cause of both conditions. Either edema, congestion, inflammation, chronic thickening, ulceration, tumor, foreign body or cicatricial contraction may be the active agent in damaging the vocal function.

Paralysis of the various laryngeal muscles produces different types of dysphonia and aphonia. Hoarseness is especially common as the result of unilateral abductor paralysis resulting from pressure upon the recurrent nerve in aneurism, etc., and from paralysis of the cricothyroid muscles resulting from lesions of the superior laryngeal nerves, as in bulbar paralysis and diphtheritic neuritis.

Aphonia results if all the muscles of the larynx be paralyzed, as in bulbar paralysis, or if the abductors be so affected, as in hysteria. The absence of aphonia and dysphonia does not assure us of the absence of serious laryngeal disease, since under these conditions bilateral abductor paralysis causes inspiratory dyspnea and stridor, and may even require operative measures to prevent suffocation.

The voice is greatly modified by changes in the structure and function of the parts above the larynx. If the soft palate be destroyed, as in syphilitic ulceration, or paralyzed, as in diphtheritic neuritis, the open nasal tone is heard, while if the nasal passages be closed by adenoids or other growth we have the closed nasal voice. Paralysis of the lips prevents proper pronunciation of the labials, and the varying types of anarthria develop in paralysis or

paresis of the soft palate, tongue, and facial muscles. Great swelling of the soft parts, absence of teeth or imperfect and irregular action of the muscles involved, as in chorea, paralysis agitans and general paralysis cause more or less characteristic types of imperfect speech.

Aphasia will be considered under the diseases of the nervous system.

## SECTION VII

### PHYSICAL DIAGNOSIS

#### THE CHEST

##### REGIONAL ANATOMY

THE different portions of the chest and location of the contained organs may best be defined by reference to the bony landmarks—the vertebræ, sternum, clavicles, scapulæ, and ribs. By such reference to the mid-sternal line, the edges of the sternum, the angle of the sternum, the epigastric fossa, the anterior, middle, and posterior axillary lines and the borders and angles of the scapulæ, sufficient definiteness may be attained without the use of arbitrary lines and terms. The location of the apex-beat or other notable feature of the cardiac examination is best accomplished by measurement from one of the sternal lines with a statement of the rib or interspace involved. The uncertainties as to the position of the mamillary line, especially in women, have properly led to a general abandonment of its use as a landmark.

It is necessary to establish some relationship, more or less arbitrary, and subject to much modification according to the character of the percussion methods employed by the examiner, between the position of the heart, lungs, liver, and stomach, and the chest wall. These relationships must be carefully memorized in a general way, but with a realization of the fact that great exactness cannot be obtained until long experience develops in the examiner a well-balanced judgment in these matters, and a good technique.

##### INSPECTION

The chest should be first carefully inspected in good light, and turned, if necessary, for the detection of pulsation or of projections from the surface. We should note the size, symmetry, shape, and color of the chest, its mobility or otherwise, cardiac and vascular

movements, normal and abnormal, and the presence of deformities, growths or enlarged glands. The rounded deep chest of emphysema (barrel chest), the slim, flat or paralytic chest of the typical pulmonary consumptive, the beaded chest of the rachitic subject, the pigeon breast of the patient who has or has had nasal obstruction, the funnel breast, the unilaterally enlarged chest of pleural effusion or pneumothorax, the asymmetry with enlargement of the left side in heart disease, and the "flared" chest may be noted. The latter is commonly associated with "Harrison's groove," running from the lower end of the sternum outward and downward along the line of diaphragmatic attachment. It develops in ill-nourished and "pot-bellied" babies, especially if respiratory obstruction be also present, and may persist into adult life.

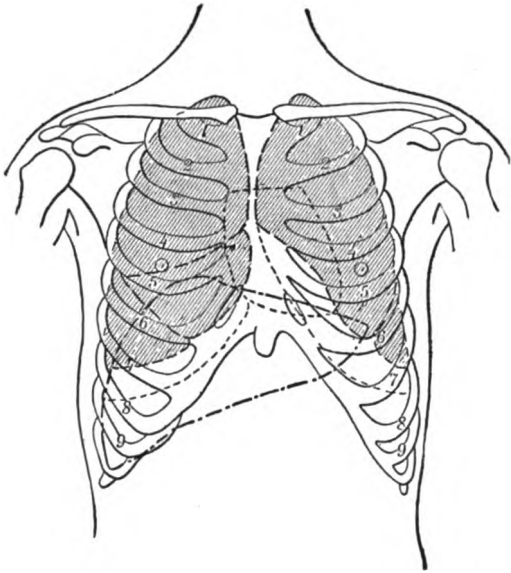


FIG. 7.—RELATION OF LUNGS, PLEURA, HEART AND LIVER TO BONY THORAX. (From Le Fevre.)

Flattening of the intercostal spaces is seen in emphysema, pneumothorax and pleuritic effusions, and tumors. In empyema necessitatis the spaces may bulge outward, and they may be seen to bulge in certain conditions upon coughing.

Prominences of the chest wall may be due to intrathoracic tumor, pulmonary hernia, aneurism, periostitis of the ribs or sternum, especially from tuberculosis and typhoid, empyema, overuse of one side of the chest, from spinal deformity, or previous disease of one lung (compensatory emphysema), protrusion of leukemic, pseudoleukemic or malignant glands, and the cardiac bulging already noted.

The chest undergoes abnormal respiratory movement in dyspnea

and excitement. The movement is generally decreased in asthma and emphysema, and locally in case of pleuritic or pericardial effusion, pneumothorax, or tumor within the chest. Diminution of the size of one side of the chest is occasionally seen in hemiplegic children, and as the result of slow contractile processes following the absorption of pleuritic effusion or associated with fibroid phthisis.

Normal and abnormal cardiac and vascular pulsations will be referred to later.

Modifications in the color of the chest wall may be either a part of the general change affecting the entire body, or localized changes.



FIG. 8.—MARKED FUNNEL-BREAST. Note egg standing upright beneath ruler.

Most important are the redness seen over a region in which pus is approaching the surface, usually associated with local edema, and of the utmost significance; the local vascular changes commonly marked by the blue color of the enlarged veins, seen along the line of attachment of the diaphragm in emphysema; and over the sternum and to some extent the sides of the chest, in obstruction of either the superior or inferior cava, the veins enlarging and assuming a deeper color as a part of their function in establishing a collateral circulation.

Over aneurismal or other tumors local discoloration may be noted.

### RÖNTGENOLOGIC EXAMINATION

"The Röntgenologic examination of the thoracic content is made by one or more of three methods. The least important of these is fluoroscopy, giving a rough idea of general conditions, but not accurate as to finer details, and not giving a permanent record. It is of value in observing changes in the air-content of the lung during expiration and inspiration, and in showing abnormal movement of the diaphragm. Of next greater importance is the study of the single Röntgenographic plate, and of the highest value, the study of the stereoscopic pair, as these give full perspective, with a separation of the mass of detail into its component parts. Exposures must be instantaneous.

"As the image upon the plate depends upon the position, form, and density of the substances giving rise to it, one will rationally be able to translate the Röntgenographic appearances upon the plate into terms of anatomy or pathology."—STOVER.

### AUSCULTATION

By this term we mean the act of listening, either with ear applied to the chest, immediate auscultation, or by means of a stethoscope, mediate auscultation, to the sounds produced in health or disease within the patient's body.

Immediate auscultation has the advantages of requiring no instrumental assistance, of giving general information as to the patient's condition in a very brief time, and of permitting certain slight and well-diffused sounds, inaudible with the stethoscope, to be heard. I have seen several cases of aortic leakage, in which the soft diastolic murmur could not be heard with the stethoscope, but was distinctly audible with the naked ear and with the phonendoscope. The latter methods of investigation apparently gathered up waves of sound from a considerable area, so that they became audible while the stethoscope could not do this. I believe every physician should habitually use the unaided ear to a sufficient extent to keep well in practice, and so that it may serve as a check upon the use of the stethoscope. In the regions about the clavicles, in separating the different heart sounds, in examining female patients, children with contagious



diseases, and in unclean patients the stethoscope offers great advantages.

In America, the binaural stethoscope is almost exclusively employed. It is important that the ear pieces fit the ear tightly yet comfortably; that the chest piece be small enough to fit well into the hollows of the chest; that the tubes be flexible yet not too easily collapsible; and that nothing connected with the instrument should be noisy or loose. The Bowles stethoscope, with the combination of ordinary chest piece and diaphragm, either of which may be selected for especial use, offers practically all the advantages of the stethoscope and the phonendoscope.

The musical twang caused by the breath impinging upon the rubber band often used, the false friction sound often caused by slipping of the bell upon the skin, the creaking of the hinge joint, the admission of external sounds through a crack in the rubber tube or in the chest piece, the friction of the tube upon the lapel of the coat, or the sudden cutting off of sound by kinking of the tube should all be guarded against as extrinsic phenomena interfering with the investigation of sounds proceeding from within the chest. An extremely dry, scaly skin, or, more especially, one covered thickly with hair, is likely to produce such crackling sounds as to prevent the detection of fine rales beneath. By greasing or wetting the skin good results may be obtained. An expert in listening with the naked ear may make a very satisfactory examination through a towel in these cases.

It requires much attention and practice to become able to devote attention to the sounds one is really listening for to the exclusion of others wholly extraneous or not being sought at the moment. It is exactly comparable to the feat of picking out and following one of the voices in a quartette, or a single instrument in an orchestra—a delicate task but perfectly feasible with training. A common error is to devote so much attention to one set of sounds as to disregard totally others of importance.

It is at times impossible to make a satisfactory examination with the stethoscope of an idiot or feeble-minded child because of the constant restless movements with such associated muscular contractions as to produce muscle sounds which obscure everything else. Patients

shivering with cold or nervousness, and athletes who breathe with unusual vigor, when asked to take a deep breath, give us the same difficulty. These indefinite rumbling or crumpling sounds should be investigated by the beginner by having them deliberately produced by contraction of a muscle while the stethoscope is applied, in order that they may not be confused with the genuine intrathoracic sounds.

When several sounds occur in very close association, as the different elements of a cardiac cycle, it is necessary to concentrate one's attention upon a single sound until it is "worked out." The confusion of sounds at first heard gradually resolves itself into a well-ordered sequence, each element of which we may appreciate and properly interpret with sufficient care and skill. When one does not hear as much as he expects over the lungs after making sure that the instrument is in good order and properly applied, he should at once consider the possibility of the presence of advanced emphysema. The exchange of air is so slight in the processes of inspiration and expiration that, if rales be absent one gets the impression from the feebleness of the sounds heard that there must be some fault in hearing them. The patient should breathe easily and somewhat deeper and faster than normal to give the best results. If in a constrained position one side of the chest may act so inefficiently as to give too feeble respiration, and even conceal rales audible with proper condition of respiration or muscular contractions may result in the production of muscular sounds which cause confusion. If the patient lies upon one side the respiratory murmur is often so enfeebled as to defeat the purpose of the examination.

In crying children it is impossible to hear much excepting at the instant of inspiration, but the conditions are nearly perfect during that brief interval. One can be sure of good results by applying the well-trained ear directly to the chest with only an intervening garment.

### PERCUSSION

We judge of the solidity or otherwise of objects in part by noting the character of the vibrations set up upon striking them. The carpenter percusses the wall to learn the location of the studding, and in so doing uses exactly the same principles that the physician does

to learn the location of the liver by percussing the chest wall with the fingers. The carpenter uses immediate percussion, however, striking the object directly, while most of our percussion is mediate, a finger of the left hand being placed over the region to be investigated, and struck with one or more fingers of the right hand or, by some physicians, with a suitable hammer.

Although it is of great advantage to be able to perceive the pitch of the sounds produced, the best results are obtained by those who study as well the quality of the note obtained in percussion, and the feeling of resistance or resiliency imparted to the percussed finger. With the ears stopped so as to enable one to better concentrate his attention upon the sensations in the fingers, one can easily note the great intensity of the resistance in percussing a solid block of wood, for example, by the feeling in the tip of the percussing finger or by noting the solidity of the blow as felt in the percussed one. It is possible for the sounds produced in percussing over a pleural effusion to approach in some degree those from a tense pneumothorax, but a different feeling of resistance to the finger can be perceived.

Immediate percussion has its chief application in examining the clavicles and the upper sternal region. I believe better results can be obtained in these two regions, if fat does not interfere, by this method.

Mediate percussion is best done with the fingers excepting in certain special cases. I have never seen any results obtained with the hammer and pleximeter that could not at least be equalled by skillful use of the fingers. If the tip of the middle finger becomes cracked or tender from excessive use, especially in cold weather, in demonstrating to students, either two fingers may be used, with some sacrifice of delicacy, or one may educate the ring finger, as I have done, and attain practically as good results, excepting for deep, hard percussion.

The pleximeter finger should be pressed firmly in contact with the part to be examined and should be struck a quick, perpendicular blow with the sharply bent hammer finger, the hand being freely movable at the wrist. The dampening effect upon the vibrations produced by anything more than instant contact of the striking finger

should be noted and carefully avoided. The unused portions of the left hand should not touch the chest wall for a similar reason.

The force of the blow depends upon the conditions as to thickness of chest wall, nearness to the surface of the organ percussed, and the object of the procedure. If, for instance, one wishes to outline for his own benefit the extreme left border of relative cardiac dullness in a thin person, the lightest percussion is to be used. If the flatness of an enlarged spleen in a fat patient is to be demonstrated to students, heavy, firm percussion, often with two or three fingers, will be necessary. In a general way we may say that the better the operator the lighter percussion stroke he will use for most of his work.

Symmetrical parts should be percussed under similar conditions, as to position, support, and muscular relaxation. One cannot percuss well a perpendicular surface much lower than the level of his own axillæ, because of the constrained position of the hands. A revolving stool upon which the patient may easily be turned and elevated is therefore of service in the examining room. For percussing the back, the patient should lean forward and draw the scapulæ apart, by placing each hand upon the opposite shoulder tip, in order to uncover the interscapular region. In percussing the axillary regions his hands should be clasped above the head. Since the skin is somewhat elevated by this procedure, any marks already made should be verified after the new position is assumed.

In outlining cardiac, splenic, or liver dullness, the examiner devotes his attention to the side involved. In comparing the lungs to ascertain slight unilateral impairment of resonance, as in early tuberculosis, accurate comparison of exactly symmetrical parts should be made, as slight changes cannot ordinarily be detected by other methods.

Certain teachers in the Vienna school and in Germany have laid much stress upon the advantages of palpatory percussion. The real object is to *feel* the resistance of the organ percussed, as well as to hear any modifications of the resonance it produces. I have seen no results at the hands of some of its most important advocates that could not be equalled by the simpler percussion already familiar to all physicians. In a certain sense we all use palpatory percussion,

since, for example, we note the increased resistance over a pleural effusion as well as the flat note.

Auscultatory percussion is of especial value in outlining the borders of the stomach. In probably more than half the cases this can be readily done with simple percussion, but in many cases colonic resonance so closely approaches that of the partially distended stomach that I always verify the results of the first method by the use of the second unless the intestinal note is very different when compared with that of the stomach. One must percuss at points equally distant from the location of the bell of the stethoscope to avoid the error introduced by variation in distance through which sounds must be transmitted. This variation may well be greater than that due to difference in the percussion notes of the different organs examined. I have long used immediate percussion with the tip of a lead pencil, used as one would use a small hammer, and am able to obtain better results than with finger percussion or the brushing motion of the finger tip often employed.

In general we may say of percussion methods that there are many good ones, but that the best results are probably to be obtained by learning thoroughly the ordinary finger percussion and then supplementing this method by such use, under special circumstances, of the other methods mentioned as may seem to add anything to the results already obtained. In my opinion, certainly ninety per cent of all obtainable knowledge of percussion phenomena may be obtained by an expert with the ordinary finger percussion.

The most delicate ordinary test of one's ability in percussion lies in outlining the area of splenic dulness in the lower left axillar region. If the spleen be not enlarged only the lightest stroke may be used, for forcible percussion brings out the tympanitic resonance of the stomach and bowel beneath. It is best to fortify one's opinion that the spleen is enlarged after percussing out its area by palpating its edge upon deep inspiration when possible.

In outlining any flat or dull area it is best to percuss at first lightly inward from the area of resonance toward the center of the dull area and mark the point of the first appreciable impairment of full vesicular resonance with the pencil. A line formed by making several such points around the dull area should give an accurate

outline of it. It is best to pay no attention to the previously made marks until all are placed, since in this way, the curve of a liver abscess projecting upward, or of an aneurism in the upper chest, etc., is much more likely to be appreciated.

**Tympanitic Resonance.**—Typical tympany is observed upon percussing a hollow viscus, as the stomach or colon moderately filled with air or gas. If gas be under great tension, as especially occurs in rare cases of valvular pneumothorax, the note may be a dull one, and fluid has been thought to be present in some of these cases. I believe the note is never the same as the flat note of effusion, nor is the palpable resistance so great in the performance of percussion.

The tympanitic note is typically high, resonant and of a peculiar quality, totally different from vesicular resonance and from flatness. The quality of the note depends upon the size and shape of the cavity containing the air, and the tension. The variation in quality may be extraordinarily great.

## THE HEART

This organ is more readily located than others in the chest because of its visible and palpable apex-beat. This is normally in the fifth interspace three and one half inches (8 or 9 cm.) to the left of the central line. The base is found at the second space or top of the third rib; the left border follows an outwardly curved line connecting the point of the apex-beat and the junction of the second left costal cartilage with the sternum; the right border is covered by the thin edge of the right lung and lies slightly to the right of the right edge of the sternum. Only a portion of the right ventricle comes in contact with the chest wall, the remainder being covered by lung. In emphysema the lung encroaches on the uncovered space and also depresses the heart, while in the shrinking of the lung, taking place in tuberculosis and fibroid phthisis, the heart is uncovered to a greater or less extent, most commonly and strikingly, owing to anatomical relations, in the region of the origin of the pulmonary artery—or at the upper left-hand margin.

The aortic valve is most readily heard in most cases at the second right interspace, next to the sternum, although not infrequently lower.

The pulmonary sound is heard less regularly at the second left interspace, but in a considerable percentage of normal individuals in the space below. The mitral sounds are best heard just within the point of the apex-beat, while the tricuspid valve is heard at a point indicated by the junction of the fifth ribs and the sternum.

The actual location of the valves beneath the surface of the chest is shown in Figure 9. The basic sounds are heard at the points above indicated rather than over the actual location of the valves, because of being propagated along the line of the blood current. The mitral

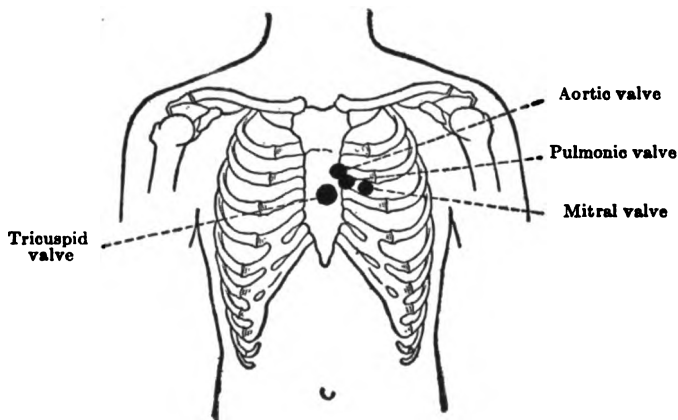


FIG. 9.—ANATOMICAL POSITION OF THE CARDIAC VALVES. (Cabot.)

and tricuspid valves are best heard near the apices of their respective ventricles if these cavities be normal in size and position. Individual variations are so frequent, however, that each case must be investigated by itself.

### PERCUSSION

The area of absolute cardiac dulness or superficial dulness corresponds with the area not covered by lung, while the area of relative dulness includes that in which the deeper borders of the heart are prevented from giving flatness upon percussion by the interposition of the overlapping lung border.

Increase of the size of the heart normally causes increase in both these areas, since the lung is pushed backward over a greater

area by the larger heart, increasing the absolute dulness, while the relative dulness is increased proportionately. The former area is easier to outline, but gives less accurate results than percussion of the deep or relative cardiac dulness. In emphysema, for example, the heart may be actually enlarged and yet show a smaller superficial area of dulness because of the pathologically distended air cells of the lung border. Old adhesions may have attached the lung border so that it is not changed by increase in the size of the heart. It is better to strive to become expert enough to depend upon an estimation of the deep cardiac dulness in judging the size of the heart.

The best results, as judged clinically, as well as by comparison with post-mortem conditions and X-ray findings, that I have seen attained, have been arrived at by the use of light percussion, although some clinicians avail themselves of the greater penetrative power of heavy percussion.

**Areas of Cardiac Dulness.**—The portion of the heart uncovered by lung gives flatness upon percussion, while a greater surrounding area gives only relative dulness because of the dampening effect of the edge of the lung interposed. The uncovered and therefore flat area has its apex at the line of junction of the fourth costal cartilages at the center of the sternum; the right border follows directly downward from this point while the left runs from the point first given to the region of the apex-beat. The lower border is fused with the flatness of the liver. This triangle of flatness increases enormously in extensive dilatation of the heart.

The area of relative or deep cardiac dulness surrounds the superficial area described, and coincides with the remaining portions of the heart so far as they can be located by percussion. The relative dulness is much more difficult of definition because of shading off into the full resonance of the lung. It gives us much more important data as to the size, power, and action of the heart than the superficial area.

**The Cardiac Impulse.**—In the fifth interspace, about  $3\frac{1}{2}$  inches from the center of the sternum and generally a finger's breadth within the nipple line, may be seen in the normal chest in most individuals an outward movement of the chest wall at the instant of



ventricular systole. This *apex-beat* or impulse marks the point of impact of that part of the right ventricle furthest downward and to the left which actually strikes the chest wall during the cardiac contraction. Percussion shows the relative cardiac dulness to be about a finger's breadth to the left of the visible and palpable apex-beat.

In children, in adults with protuberant abdomen, and in the recumbent posture, the beat may be located in the fourth space. In the upright position, especially beyond the fifth decade, the sixth space may show the point of maximum impulse, but such a location ordinarily raises a presumption of disease. If the chest wall be over fat, and the heart's action unusually quiet or enfeebled by disease, the beat may be impalpable, or even invisible. Changes in lateral position are to be noted through assuming the left lateral posture, less marked in the right lateral position. The vertical position is lowered slightly by deep inspiration and raised by abdominal conditions preventing a normal descent of the diaphragm. The apex-beat is commonly displaced to the left under these conditions.

The influence of emphysema in crowding the heart downward and inward and concealing the normal apex-beat, and at the same time giving rise to epigastric pulsation through impact of the displaced right ventricle, should be noted.

When the heart through enlargement, either by hypertrophy or dilatation, or through retraction of the lung borders because of contracting processes in the lung or pleura, comes in contact with a greater area of the chest than normal, the whole precordial area may heave with the heart beat. Violent action of the normal heart, especially in children, may produce a similar impulse.

A wavy undulation seen across the precordial area is generally significant of dilatation and weakness of the heart.

Investigation of the precordial area and the position and character of the cardiac impulse is much interfered with by obesity and especially by the presence of large mammae. The left breast may so cover the heart as to prevent accurate localization by percussion and to interfere very seriously with auscultation.

**DISPLACEMENT OF THE CARDIAC IMPULSE.**—The examiner

should accustom himself to locating the region of cardiac impulse at the first glance of the chest, so that any abnormality in location or area or any extra pulsation will attract immediate attention.

The most common displacement is toward the left, since the most common condition causing displacement is mitral regurgitation, which causes secondary dilatation and hypertrophy of the right ventricle, and consequent increase in the transverse dimension of the heart, with displacement of the apex-beat to the left. Since the left ventricle is also involved, with increase in its length, some downward displacement is commonly present. In general, displacement to the left is due to right-sided cardiac disease, while left-sided disease, especially aortic regurgitation, causes the apex-beat to be located farther downward.

Probably the next most common cause of displacement of the apex-beat and the cardiac area is pulmonary tuberculosis. Since one lung is ordinarily involved to a considerable extent before the other shows notable signs of disease, the contractile processes incident to cavity formation, fibroid changes and pleural involvement, in association with the compensatory emphysema in the opposite lung, lead to marked displacement. A lesser involvement is necessary to produce extensive changes upon the left side, and a more immediate tendency is noted here toward a raising of the point of cardiac impulse, since the normally suspended heart lies to the left of the "dead center" of suspension.

Other fibroid conditions of the lung and contracting processes in the pleura, especially following effusion, bring about similar dislocation. Displacement after effusion, however, raises no presumption as to the side upon which the effusion took place, since the heart may have been pushed over and become adherent, or may have remained unattached and thus been drawn back beyond the normal location by a secondary contracting process.

Chronic fibroid disease of the right apex may displace the cardiac beat even to the right anterior axillary line, and draw it upward to the fourth space.

Displacement by effusion in one of the pleural cavities or by pneumothorax, should be mentioned as the next cause in order of

frequency. A lesser accumulation of fluid or air on the left side suffices to displace the heart to the right than is requisite on the right to displace it in the contrary direction. When passing beneath the sternum in its journey toward the right in extensive left-sided effusion, the apex-beat cannot be located.

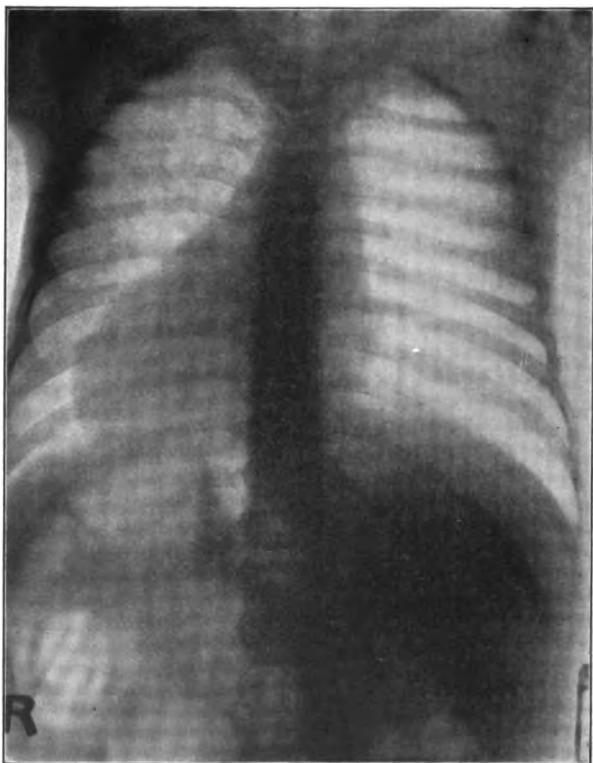


FIG. 10.—TRANSPOSITION OF THE VISCERA IN A BOY 12 YEARS OLD. Anteroposterior view. (Dr. S. B. Childs.)

In complete situs inversus, in which all the viscera are transposed, the heart is placed symmetrically upon the right side as it should normally be upon the left. Congenital dextrocardia alone is very much less common, and should be diagnosed only after most searching inquiry as to previous lung or pleural disease. I have found five cases of complete situs inversus and seen one at post mortem in twenty years, but have never seen congenital dextrocardia

to my knowledge. By accurate percussion of the heart, liver and spleen, study of the artificially dilated stomach, determination by digital examination of the course of the rectum upward toward the right instead of toward the left as usual, the use of the X-ray, and in males, observation of the lower position of the right testicle instead of the left, as is normally seen, there should be no reasonable doubt as to the correctness of the diagnosis of transposition of the viscera. In one case I found additional evidence associated with the presence of an apical systolic murmur. This was transmitted toward the right and accompanied by accentuation of the basic sound in the second right interspace. This would be possible only in a symmetrical transposition of the heart with the pulmonary artery upon the right side of the chest.

In lateral spinal curvature, kyphosis, and in pigeon-breast, various displacements of the heart occur. In marked funnel breast I have found the heart pushed to the left by the inward projection of the chest wall.

Downward displacement of the heart may occur with the sagging of the aorta seen in old arteriosclerotic individuals. Aneurism of the aorta, mediastinal tumors, and enlargement of the mediastinal glands from tuberculosis, pseudoleukemia and other causes, may depress the heart, generally toward the left.

**RETRACTION OF THE APEX REGION.**—The sinking in of the area around the point of apex impulse due to violent action of a hypertrophied heart (with its consequent negative intrathoracic pressure) has no especial significance. Much more important is the positive systolic retraction brought about by complete pericardial adhesion, so that with the cardiac contraction the fleshy part of the chest wall is both seen and felt to sink inwards. This is a most important sign of adherent pericardium.

**Abnormal Areas of Pulsation.**—One should think first of uncovering of the heart by retraction of the thin border of the lung already described. The forcible, visible and palpable “snap” of the pulmonary valves working under increased tension when the base of the pulmonary artery is brought closer to the chest wall by retraction of lung from tuberculosis of the left apex, is a very common

clinical phenomenon. In chlorosis, exophthalmic goiter and other debilitating diseases less prominent pulsation is seen. Aneurismal pulsation is commonly at such a distance above the apex-beat as to be readily recognized. A lateral illumination of the chest wall is of assistance in many cases. Pulsation in the suprasternal notch from aneurism, dilated aorta or goiter may be noted. The pulsations of a long neglected purulent pleurisy, which has eventually started to burrow through the chest wall, are almost always in the upper portion of the left chest. They are synchronous with the heart beat, and may appear in the recumbent posture. Inflammatory edema is often present over the pulsating area and aids in the diagnosis.

The movement of the left lobe of the liver transmitted from the violent action of the hypertrophied heart, and the venous pulsation of the liver in marked tricuspid regurgitation, should be noted.

**Effect of Pericardial Effusion.**—The accumulation of fluid in the pericardial sac, whether this be hemorrhagic, inflammatory or dropsical, results in an extension of the area of precordial dulness. Because the fluid naturally gravitates to the lower portions of the cavity, the dull area is broader below, and extends to the right and left much beyond the usual lines of heart dulness. The heart tends to retreat somewhat from the chest wall in case of great effusion, so that the apex-beat is either not felt or felt but feebly, and in the latter case oftentimes higher and slightly more to the left than normally, but still well within the limits of dulness. The presence of flatness to the right of the sternum, broadening the cardiohepatic angle, is fairly reliable evidence of pericardial effusion. The muffled sounds of the heart, embarrassed by the effusion, and the presence in the upper cardiac area of pericardial friction sounds, may help us to the diagnosis. The intercostal spaces may be obliterated by being filled out through the presence of the fluid within.

The tympanitic resonance in the upper left chest in front, and the impaired resonance in the left scapular region seen in large effusions should be noted. Yet I have seen both these signs in children with very large hearts. It has sometimes been impossible for the best diagnosticians with whom I have been associated to distinguish between pericardial effusion and great dilatation of the heart;

and I have seen more than once the aspirating needle bring pure blood from the dilated cardiac cavity instead of the serum expected, and with even more striking relief than the latter procedure usually gives.

In the largest effusions there is but little lateral movement of the area of dulness upon change of position, and this chiefly toward the right side. The apex-beat may be more easily felt with the patient in the right lateral position in case of pericardial effusion, while the contrary holds true in dilated heart. The pressure symptoms of large pericardial effusion are at times of much importance in the study of cyanosis, paroxysmal dyspnea, cough, aphonia and dysphagia.

In very large effusions the raising of the clavicle permits one to feel the upper edge of the first left rib. (Ewart).

Pleuritic effusion, especially if encapsulated, may be very difficult to differentiate, even after pus has been obtained by aspiration within the cardiac area. I was in doubt in one case\* whether it did not come from encapsulated empyema until after resection of a rib.

In rheumatism we may have both pleural and pericardial effusion present and one may remain unrecognized until its characteristic features are brought out by the removal of the fluid of the other effusion by aspiration. It is stated that the pericardial effusion is more commonly overlooked than any other gross lesion within the chest.

**DRY PERICARDITIS.**—Owing to inflammation of the serous surface of the pericardium roughening occurs, giving the dry form of pericarditis. A plastic exudate occurs in many cases, later, a fluid effusion. The inflammation causing it may be rheumatic in origin, may originate by extension from pleuropneumonia, especially on the left side, may be the result of trauma, as from a fish bone in the esophagus, or may be due to tuberculosis, cancer, gumma, etc.

**PLASTIC PERICARDITIS.**—The rubbing of the roughened pericardial surfaces with the cardiac movements gives rise to the only sign by which we may recognize plastic pericarditis, namely—pericardial friction. In many cases of pneumonia and doubtless when pericarditis occurs in other connections, the friction sound is present for but a few hours, the occurrence of effusion, or the “gluing” together of

\* Reported by Gengenbach.

the roughened plastic surfaces causing it to disappear. We may thus account for the well-known fact that plastic pericarditis is so frequently found post mortem when unsuspected during life.

Excepting for the increased frequency of heart beat, which, with the forcible action, may generally be attributed, in part at least, to the febrile affection with which pericarditis so frequently occurs, we find no evidence of the presence of plastic pericarditis aside from the palpable or audible friction. This friction is best felt with the fingertips placed in the interspaces over the region in which the heart comes in contact with the chest wall. If unusually evident, however, the flat hand laid over the precordia perceives it readily. The rhythm is generally systolic, but often diastolic as well—a to-and-fro rhythm. It gives the impression of two roughened surfaces brushing by each other with a light touch.

Upon auscultation the friction sound seems closer to the surface than do the intracardiac murmurs. Generally systolic in time whenever present, it is also often diastolic but less accurately synchronous with the phases of the heart's action than are the valvular murmurs. Varying the pressure with the bell of the stethoscope often changes the character and intensity of the sound. The cessation of respiration has no effect excepting to make the pericardial friction more clearly distinguishable. Inspiration and expiration often change the character of the friction but in no constant and definite manner.

The sounds are best heard over the uncovered portion of the heart, or area of absolute dulness, but are often present elsewhere as well. As effusion occurs and separates the visceral layer of the pericardium from the parietal, friction may be heard only in the region of the base. If the patient leans far forward the heart may come into more intimate contact with the chest wall and intensify the friction sounds.

Pericardial friction may be accompanied with or confused with pleuropericardial friction. This is most often heard at the lower left border of the heart. The surfaces involved are the two layers of the pleura lying superficially and those of the pericardium more deeply situated, and either pair may be affected by an inflammation extending either outward from the heart or inward from the lung.

It is impossible to be sure of the differential diagnosis in certain cases.

If the friction murmur over the heart ceases with respiration we may be sure it is produced by the pleural surfaces. Pericardial friction generally is best heard when the lungs are most retracted from over the heart, that is, at the end of expiration. At this very time the pleural friction often disappears, at least from the cardiac area.

**ADHERENT PERICARDITIS.**—As the result of a slow obliterative inflammatory process the layers of the pericardium become adherent and the pericardial cavity disappears in full, or in part. In children this process often accompanies extensive valvular disease, and marked prominence of the precordial area is noted. So common is failure of compensation in children of this type that it may generally be assumed that failure of compensation with great prominence of the left ribs and dwarfing of the figure is due even more to the embarrassment of the heart from the adhesions than to the valvular defect. The chest may, however, be flattened. The systolic retraction in the region of the apex-beat has been mentioned. Broadbent described the retraction seen in the lower left back, 11th and 12th spaces, noted especially in deep inspiration. The heart in contraction pulls upon the neighboring structures because of the adhesions to the parietal layer of the pericardium and its attachments. The respiratory movement is decreased below the precordial region near the ensiform cartilage and especially if mediastinal adhesions be associated. A diastolic rebound of the cardiac apex is often to be noted, especially if the union between the pericardium and the chest wall be a firm one.

Friedreich described the collapse of the cervical veins during diastole, but it is probably not as frequent nor as reliable a sign as formerly believed.

Since the adhesions tend to fix the heart it does not shift in its position as readily as is normal, either laterally or vertically. Respiratory movements of the lung border out over the cardiac area of dulness in deep inspiration are nearly or quite abolished if extensive adhesions are present. The same failure of movement is noted, especially at the upper left margin of the heart area, as the result of



the pleural adhesions of tuberculosis, and the two factors may work together in certain cases.

Adherent pericarditis may occur as a part of a disease process in which the pleura and peritoneum are also involved, and a chronic mediastinitis occurs. Marked circulatory disturbances follow, with enlargement of the liver and ascites. It is probable that the mediastinitis is the chief cause of the trouble, but its presence must be inferred from the signs and symptoms of the pericarditis with which it is associated. A search for these should be suggested by the occurrence of enlarged liver and ascites not otherwise explicable. The distortion of the outline of the heart's shadow as shown by the Röntgen ray is of value in the diagnosis.

The right heart, from its more extensive area of possible attachment anteriorly, is more likely to be compromised than the left.

### PALPATION

**The Apex-beat.**—This is best found by laying the palm on the chest below the left nipple. The normal heart gives a regular firm "tap" which may be investigated more closely by palpation with the tip of the finger. It is generally found a finger's breadth within the nipple line, or  $3\frac{1}{2}$  inches to the left of the center of the sternum and in the fifth interspace. A lighter beat may be felt with care at the extreme edge of cardiac dullness.

If the heart be much hypertrophied, as in chronic nephritis, we feel it heave under the hand, as if it lifted the whole chest wall. In palpitation and in nervous action seen in exophthalmic goiter it strikes forcibly and rapidly but without the heave of hypertrophy. In obese persons the beat may be imperceptible though the heart action is presumably normal. In those with advanced arteriosclerotic types of myocarditis, the action is so feeble that often it cannot be palpated. The widely dilated heart gives a feeble "slap," the beat being often better seen than felt. In mitral stenosis, the beat is sudden and forcible, giving to the sense of touch much the same impression that the sound does to that of hearing. In aortic stenosis the slow deliberate contraction of the left ventricle gives a thrust rather than a beat.

The heart beat may be felt more distinctly in those positions permitting it to strike most directly upon the chest wall. Since it changes position laterally with corresponding movement of the body to a distance of an inch or more we may find it less easily palpated because of being placed further under the covering edge of the left lung or more hidden under a rib or the costal cartilage, in varying positions.

At the base of the heart we may often feel the diastolic shock of closure of the basic valves, though often indistinctly because of the covering lung border. The sharp shock of the pulmonary valves when the left lung is retracted in fibroid phthisis and the pulmonic pressure is increased under the influence of the consequent hypertrophy of the right ventricle, is very characteristic.

**Thrill.**—Over the region of the apex most commonly, but not rarely elsewhere, we may obtain the vibratory sensation described as a thrill. It imparts to the palpating fingers a sensation similar to that felt when a saw is drawn across a piece of wood or a file across metal. The back of a purring cat gives much the same sensation to the hand. The thrill is confined usually to a small area near the valve at which originate the vibrations, which, communicated to the chest wall, produce the phenomenon we describe.

For the production of thrill it is necessary that a blood current pass with force through an orifice narrowed, roughened, or distorted. Since the more powerful muscle walls of the cavities of the left side of the heart produce a higher blood tension than those of the right, the thrill is most frequently found at the mitral and aortic orifices.

The thrill is practically pathognomonic of certain cardiac affections. When present in valvular disease it necessarily occurs in a single phase of the heart action and signifies obstruction to the current passing through that phase. If double thrill exists, as is occasionally the case, the systolic and diastolic elements are readily separated.

While more constant than many cardiac murmurs, thrills depend so closely upon the force and tension of the blood stream that they may appear and disappear with changes of the heart strength. This applies especially to that of mitral stenosis, for the thrill may dis-

appear with temporary cardiac exhaustion, and with rest and digitalis reappear in a few days. The disappearance is thus of evil significance as indicating a break in compensation.

The most common thrill is that at the apex in mitral stenosis—a presystolic thrill caused by the passage of blood through a narrowed and roughened mitral valve, under the impulsion of the hypertrophied left auricle. Much less frequent is the systolic thrill of mitral regurgitation. The two may occur in the same case.

Thrills due to aortic disease are much less frequent than those at the apex, and are systolic or diastolic according as they originate with the forward passage of the blood stream, or with regurgitation. The diastolic thrill is of more value than the systolic as indicating necessarily a valvular leakage of importance, while the systolic thrill may be due to roughening without great obstruction, and perhaps to hemic conditions. It is generally propagated into the vessels of the neck.

The only thrill I have observed at the tricuspid valves was presystolic in character and due, of course, to the rare tricuspid stenosis. Thrill existed at the mitral region, and marked obstruction was found at both orifices post mortem. Systolic thrill from tricuspid leakage must be rare, but not at all impossible.

Systolic thrill is not rare in congenital pulmonary stenosis and I have once noted diastolic thrill associated with congenital deformity of this valve, which permitted leakage. No very accurate deductions can be drawn as to the cause of thrills at this orifice, however, since congenital deformity of the conus arteriosus, valves, ductus arteriosus, etc., may so complicate the situation as to make ante-mortem diagnosis very uncertain. I have seen continuous thrill reinforced with systole in congenital heart disease.

Thrill is not uncommon in aneurism of the aorta and is likely to be felt at the aortic or pulmonary areas and to be systolic in time. I have twice noted a continuous thrill reinforced in the systole over an extensive area near the heart, where the post mortem showed that an aneurism of the aortic arch had perforated into the pulmonary artery. The sudden development of such a thrill as this in well-defined aortic aneurism is very suggestive of the condition described.

**The Pulse.**—Because of its availability, the radial artery has long

been used in examination of the pulse. It is better to examine both vessels at once since one is often more accessible or larger than the other or one gives an improper idea of the circulation because of atheroma, or gives a smaller or delayed pulse because of aneurism, pressure by tumor or other cause. For counting the pulse and ascertaining its rhythm we may utilize the facial, temporal or carotid arteries, but the blood pressure can be estimated with much greater accuracy in the radial or femoral.

With the tips of two or three fingers we note the pulse wave in the vessel. It is well to ascertain before studying the pulse the condition of the arteries as to the palpability of their walls. The vessel wall cannot be distinguished in the normal artery of youth. In most individuals in middle age sufficient arteriosclerotic change has developed to render the artery palpable. It is best noted by rolling the vessel under the finger tips. If difficulty arise we may make sure of thickening of the walls if we can distinguish the vessel by rolling it beneath the tips of the finger *nails*, even though it be imperceptible to direct touch. Moderate thickening is so common after forty years of age that it is not to be regarded too seriously, for in many such cases no histological evidence of disease of the walls is to be found post mortem.

To the thickening of the intima is eventually added tortuosity of the vessel, readily seen in superficial vessels, but most easily in the radial, brachial and temporal arteries. This signifies that an actual endarteritis has begun. Increased blood tension is to be sought for, and is often found under these conditions.

To arteries such as we describe there is likely to be added a third important feature—calcification. We feel a rough “beaded” vessel wall, and in extreme cases, great thickening and tortuosity are associated with an extreme degree of calcareous degeneration. The artery feels like a pipe stem, and is so rigid as to be incompressible. In extreme cases no pulse wave can be felt in the vessel, and its arterial character is determined more by its anatomical relations than by its feeling.

Care must be observed not to confuse thickening and incompressibility of the arterial walls with increased blood tension. Our diffi-

culties are increased when both conditions are present simultaneously.

Having noted the character of the arterial wall and its location, size and tortuosity or otherwise, the degree of compressibility should be estimated by noting the pressure necessary to obliterate the pulse wave. It is well to examine the femoral as to this point and to compare the result arrived at with that shown by the blood pressure apparatus to be described later.

The rate of the pulse, varying from 120 to 140 in the infant, to the normal 60 to 80 in the adult, is determined by counting for from 10 seconds to a minute according to the degree of accuracy desirable under the circumstances of the case. The average male adult has a pulse rate of 72, the female of 80. The greatest individual variations exist and should not be considered pathological without proper evidence.

The rhythm of the pulse should be observed. Excepting as it varies slightly, especially in children with inspiration, it should normally be regular in time and force, irregularity in the latter particular being of more serious significance than lack of perfect time. Intermittence is so common, either throughout life as a congenital idiosyncrasy or occasionally as the result of overwork, loss of sleep, over-use of coffee, tobacco, etc., that it should not be taken necessarily as an indication of disease. Variations in regularity and in force are more serious, and are especially common in mitral disease. They will be further considered under valvular disease of the heart.

The characteristics of the pulse wave should be observed. The size is estimated by the "lift" of the arterial wall under the finger; the duration by the length of time that the lift is sustained; and the tension by the degree and permanency of distension of the vessel. These three elements are combined in various ways, although generally in a systematic manner. Wide dilatation of the capillaries and arterioles lessens the peripheral resistance to the circulation, and tends to produce a pulse of large size, since the wave is propagated under conditions of low tension dependent upon this very lack of peripheral resistance. From the same cause the pulse is not well sustained, or in other words, has a short duration. *Per contra*, high

peripheral resistance tends to give, of course, a pulse of high tension, of small size, and of longer duration.

The shape of the pulse wave, best determined by the sphygmograph, can yet be estimated with some degree of accuracy by the trained finger. The wording used in describing the shape of the pulse wave has apparently been derived from that used in the study of the visible image as produced by the sphygmograph. Thus we speak of a "peaked" wave, when the pulse is ill-sustained from aortic leakage, or decreased peripheral resistance, often in combination with weakened heart power. The sudden rise of the wave throws the lever of the instrument high, but the lack of sustained tension permits it to fall suddenly. Under an opposite set of conditions the wave rises slowly, is well sustained, and then falls slowly. If we suppose a peripheral arteriosclerosis with high tension and such aortic obstruction with good compensation as to make it necessary and possible for the heart to fill the aorta slowly and with great power, we should have the ideal condition for the production of a pulse wave of a "long plateau" type.

The dichrotic pulse wave so readily perceived in conditions of low tension and peripheral relaxation, as in early typhoid fever, represents an exaggerated secondary wave perceptible to the finger. It is more readily detected upon a well-taken sphygmographic tracing. If not perceptible to the finger it is of little clinical importance. The anacrotic pulse, in which this secondary wave is carried over to the ascending limb of the tracing, is seen in rapid heart action, but its study is of no great value to the physician at the bedside.

**Arterial Pressure.**—SPHYGMOMANOMETER.—Recently introduced instruments enable us to estimate with sufficient accuracy the arterial tension. The clinician is most interested in learning the height above normal of the systolic pressure, while physiologists and investigators are interested as well in the diastolic pressure. The instrument should have a wide armlet (10 cm.), as with this the varying degrees of resistance offered by larger and smaller arms and by differing conditions of the vessel walls may be practically disregarded in comparative examinations. As in taking the temperature, the physician wishes to learn *approximately* how far the conditions depart from

normal. An absolutely exact estimate of the blood pressure cannot be obtained, since it varies somewhat from moment to moment. If we can know within a few millimeters our purposes are well served.

The estimation of the minimum or diastolic pressure may be made with several types of sphygmomanometers by noting the moment of greatest oscillation of the mercury in the glass tube, by clamping the air tube after obtaining the systolic pressure and then permitting the air to escape gradually until the oscillations desired appear.

The most recent methods of obtaining systolic and diastolic pressures involve the use of the stethoscope applied to the brachial artery distal to the band. The first sound heard as the air pressure is reduced indicates the passage of the first wave through the artery, hence the systolic pressure. The time of development of the loudest "thud," as the pressure falls is probably the most dependable for the taking of the diastolic reading, although the time of disappearance of all sound, a few m.m. below the "thud," may be substituted.

The pulse pressure, indicated by the difference in m.m. between the systolic and diastolic pressures should ordinarily be about 35 per cent. of the systolic reading, or 50 per cent. of the diastolic, thus falling in normal cases between 25 and 40 m.m.

In aortic regurgitation the sudden fall of pressure in diastole may cause a doubling of these figures.

By far the most important use of this valuable addition to our means of investigating the condition of the circulation is the estimation of the systolic pressure in the great class of cases in which arteriosclerosis and kidney disease are more or less associated in affecting the arterial tension.

The normal blood pressure varies between 70 or 80 m.m. in children and 140 or 150 in adults beyond middle age. In chronic nephritis and arteriosclerosis pressures of over 300 m.m. may be found.

The temporary changes of tension due to emotional influences, violent muscular exertion, the use of drugs, etc., although often of great importance to the patient, are not of such character as to be so satisfactorily studied as are the chronic conditions mentioned. In-

crease in tension is of vastly more importance to the clinician than the contrary condition. Many excellent blood pressure instruments are now upon the market.

### AUSCULTATION

After inspection, palpation, and percussion we are in a better position to profit by auscultation of the heart sounds. Although

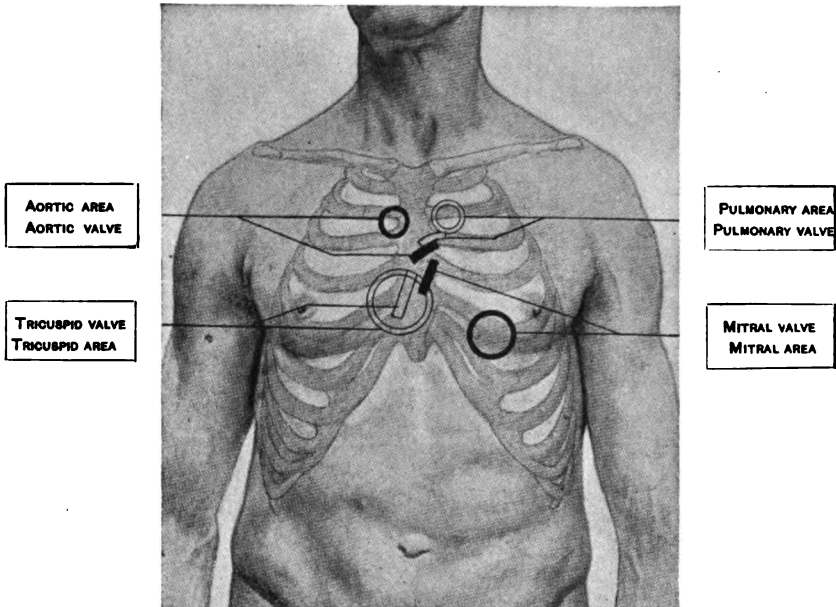


FIG. 11.—SHOWING THE POSITIONS OF THE VALVES OF THE HEART AND THE AREAS OF THEIR GREATEST AUDIBILITY. (Butler.)

with the naked ear we may often distinguish cardiac murmurs easily, the sounds, normal or otherwise, are generally better “picked out” with the stethoscope. Occasionally a soft, diffused murmur, especially one of aortic regurgitation, may be heard with the naked ear, or with the phonendoscope, when inaudible with the stethoscope, as if by the former methods we were able to gather waves of sound from a larger area than with the stethoscope.

**The Valves and Their Sound Areas.**—Figure 11 shows the anatomical position of the cardiac valves, while Figure 9 shows the



areas over which the sounds produced at the different orifices are to be best heard. Because of the direction of the blood current associated with the production of the different sounds and the transmitting power of the chest wall and the tissues of the contained organs, the two sets of areas do not correspond. The sound of the aortic valves is best heard in the second right interspace near the sternum, but the area of the maximum sound may be slightly lower. The murmurs originating here are often best heard either above or below this point, according as they are direct, transmitted upward with the blood stream, or regurgitant and transmitted downward.

In the second left space, or, more frequently than in the case of the aortic valve, in the one below, may be heard the sound of the closure of the pulmonary valve. The murmur of an obstructive lesion at this point is transmitted toward the interscapular region rather than upward, owing to the course of the pulmonary artery, while that of regurgitation is transmitted downward in much the same way as that of aortic leakage. At a point just within and below the nipple, we hear the normal first sound of the heart, this being termed the mitral area, because here we listen for either of the murmurs produced at this valve. The stenotic murmur is quite sharply localized as a rule, while the murmur of regurgitation is transmitted to the left or into the back, or upward toward the pulmonary area in some cases. Near the lower end of the sternum, perhaps most frequently upon the left side, but often on the right, is the area of the tricuspid valve. Its murmurs, direct or regurgitant, are as a rule more limited in extent than those of the mitral.

**Heart Sounds.**—The rhythm, quality, area of maximum intensity and modifications of the sounds by various agencies in health and disease are to be sought for.

The first sound of the heart, coinciding with the thrust of the apex-beat, is best heard at the mitral area, and following it we hear at the same point the second sound, coinciding with the closure of the basic valves. The first sound, commonly described as resembling that produced by pronouncing the syllable "lubb," is presumably a composite sound produced by the impact of the apex against the chest wall, by another sound due to the contraction of the muscle, and an-

other arising from the closure and tension of the mitral and tricuspid valves and their auxiliary structures.

The second sound, "dup," arises from the closure of the basic valves. Then follows the diastolic interval, normally somewhat greater than that occupied by the two heart sounds. Although the two sounds in health differ so markedly as to render differentiation by sound alone comparatively easy and certain, in disease the qualities change so markedly as to render it necessary to verify the location of the first sound in the cardiac cycle by palpation of the apex-beat, or the carotid artery. The heart weakened by disease may have much the same rhythm as the fetal heart, and we may then be in doubt whether we hear only the first sound, only the second sound, or the two, alternating but indistinguishable.

The first sound is the predominating one in the mitral area, but at the base of the heart the pulmonary or aortic sound assumes the leading place according as we listen over the left or right second interspace. It is possible, however, for either basic sound to be so accentuated in disease as to dominate the whole basic area.

The intensity of the heart sounds is modified by the vigor or feebleness of the cardiac action, and the thinness or thickness and elasticity or rigidity of the chest walls. In general, the sounds are clearer and sharper in children than in adults, and the valvular element in the first sound is more pronounced than the duller muscular sound which gives the characteristic booming quality to the adult heart beat.

The intervention of lung or other tissue between the heart and the chest wall may markedly modify the intensity of the sounds in accordance with the density of the tissue in question. The withdrawal of the thin edge of lung from over certain parts of the heart permits the sounds to be propagated to the surface of the chest with more than normal force and distinctness.

**INCREASED INTENSITY OF FIRST SOUND.**—Causes tending to increase the force of the first sound of the heart are in general those causing more vigorous action of the organ, and especially exercise and excitement. The heart, the muscular walls of which are hypertrophied by athletic training, or stimulated to increased action in the

early stages of fever, or the one whose cavities and walls are modified by the dilatation and hypertrophy of valvular disease, yields an accentuated first sound. When the vascular tension is increased, as in interstitial nephritis, the first sound has more of the booming quality associated with increased muscular work. The rapid and forcible heart action of Graves' disease, on the other hand, presents more of the valvular element. The increase in force of the first sound in well compensated mitral stenosis is recognized more easily than explained.

**INCREASED INTENSITY OF SECOND SOUND.**—The increased intensity of the second sound is due to increased pressure in either the systemic or the pulmonic circulation, with correspondingly forcible closure of the aortic or pulmonic valves, except that in rare instances the two causes may be combined. The increased force may be more apparent than real in those cases in which the valve is uncovered by the retraction of the thin lung border in lung disease. If accentuation be present for any considerable length of time it implies hypertrophy of the ventricle upon the corresponding side, such increase in size and power of the muscle being necessary to overcome the resistance which has caused the increased pressure with consequent accentuation of the valve closure.

Cabot has shown that in youth the pulmonary sound is commonly louder than the aortic, while after middle age the contrary holds true. In the middle third of life either valve may give the louder sound. Judgment should therefore not be based solely upon comparison with the other basic sound, but upon the usual force of such a sound in an individual of similar age and physique.

Arteriosclerosis and nephritis are the chief causes of aortic accentuation, while mitral disease and chronic disease of the lungs tending to narrow the pulmonary blood paths are the chief factors in increasing the sound of the pulmonic valvular closure. In event of an increase of pressure greater than the ventricle can sustain permanently, the auriculoventricular valve commonly becomes incompetent through stretching of its muscular ring, and the pressure upon the corresponding side of the circulation thereupon falls.

**WEAKENING OF THE FIRST SOUND.**—Aside from those conditions of obesity, interposition of abnormal tissue elements, as the enlarged

emphysematous lung or thickened pleura, or other mechanical condition rendering the first sound less easily audible, it may become weakened because of failure of muscular power in the heart wall. This is generally the result of some degenerative process affecting the muscle tissue, such as results from starvation, the effects of continued fever, chronic enfeeblement from lack of blood supply in atheroma of the coronary artery, etc. When the muscle sound is greatly decreased by such causes the valve sound, more "snapping" in quality, becomes plainer.

Decrease in intensity of the basic sounds arises chiefly from lowering of the blood pressure, systemic or pulmonic, or both, as the case may be. Loss of a part of the general mass of blood, as in severe hemorrhage, incomplete filling of the vessels, as in valvular disease, lack of the usual resistance in the peripheral vessels, as in Graves' disease, or damage to the structure of the valves may be a factor in lessening the pressure. Marked weakening of the basic sounds is of serious import as an indication of pronounced cardiac weakness.

In studying changes in strength of the first sound at the apex we should correlate our findings with the knowledge to be derived from palpation of the apex-beat. If contracting disease of the lung uncover the base of the heart we may feel the sharp closure of the basic valves by placing the finger in the second interspace near the sternum, much more commonly when contracting disease of the apex of the lung uncovers the conus arteriosus than upon the aortic side. Variations in pressure cover a much greater range in the aorta than in the pulmonary artery, and the sharpest ringing sound to be heard at the base is in cases involving the systemic circulation and, of course, over the aortic area.

CHANGES IN THE RHYTHM OF HEART SOUNDS.—*Embryocardia*.—This name is applied to the feeble and rapid action of the heart which develops after the prolonged action of causes tending to weaken the heart muscle. The first sound of the heart is heard with much increased frequency, this being naturally at the expense of the diastolic pause, and the heart has then the "tick-tack" sound of that of the fetus. It is significant of profound exhaustion of the heart

muscle, and is found in the course of serious febrile diseases, in loss of compensation in heart disease, and also in cases of acute collapse from various causes.

*Gallop Rhythm.*—In serious cardiac exhaustion, occasionally when this is acute in character, we may have, in conjunction with shortening of the diastolic pause, the interpolation of another sound, due to the “doubling” or “splitting” of the first or second cardiac sound, with the result that we have at the apex three sounds, often differing but little one from the other, with the rhythm of the hoof-beat of a galloping horse, or the “rub-a-dub” of a drum beat. The general impression of heart failure given by this gallop rhythm is of more import to the clinician than any particular indication derived from an exact analysis of the condition.

*Reduplication of Heart Sounds.*—Sewall has shown that splitting of the basic heart sounds is extremely common at the end of a long inspiration and under some other conditions, without any special pathological import, being due to increased pulmonary pressure and asynchronous closure of the basic valves.

French authors have long laid much stress upon doubling of the basic sounds in the diagnosis of mitral stenosis, the doubling being often audible at the apex. It has been explained as being due to premature closure of the pulmonary valve because of earlier attainment of a sufficient intraventricular pressure to cause systole in the right ventricle, with its embarrassed pulmonary circulation to carry, than occurs upon the left side. This is in dispute, some physiologists claiming that the exact opposite holds as regards the times of closure of the two valves. We may take it as settled, however, that reduplication at the base is the result of variation in the relative pressures in the two sides of the circulation.

*Other Unusual Heart Sounds.*—If the *systemic* pressure be very high, and perhaps if this be associated with atheromatous changes in the aortic valves, the aortic second sound may show a distinct metallic “clink” in closure. Under similar conditions the first sound at the apex may be of ringing, almost musical character. I believe that the pulmonary circulation never produces the counterparts of these two sounds. Occasionally in pneumothorax and occa-

sionally when the stomach or the colon is distended with gas, an almost bell-like tone is given to the first cardiac sound.

It is probable that "muffling" of the first sound generally means feebleness of the muscular element and clouding of the valvular element by leakage, not yielding the usual systolic murmur. Whatever the exact explanation, it involves feeble heart action so that the usual cardiac sounds are not clear.

**Cardiac Murmurs.**—The abnormal sounds produced within the heart are usually described as murmurs. For the passage of the blood through the valves of the heart without other than the normal sounds already discussed, it is necessary that the blood be fairly near the normal in composition, that the force of the current be approximately normal, that the valves be structurally intact, with the implied ability to open and close perfectly, and that the vessels and cavities in relation with the valves be relatively of proper size and normal function. If these various relations be materially altered, we may have the production of eddies or "fluid veins" which produce the vibrations which in turn give rise to the murmurs we study.

Murmurs are classed as organic, due to structural alterations of the valves, and inorganic, due to other causes. We will first study the latter.

**INORGANIC MURMURS.**—If every portion of heart and vessels be structurally perfect we may have murmurs, due, it is believed, to changes in the blood, especially lowering of the specific gravity, associated with anemia. These murmurs are predominantly systolic in time, and are spoken of as "hemic murmurs." They may appear in a few minutes after a severe hemorrhage, when permanent structural alterations may be safely ruled out. The disappearance of such murmurs, after, e. g., the administration of iron in chlorosis, is further proof of their inorganic nature. It is probable that increased force and rapidity of the blood current may produce murmurs which are classed as dynamic. It is likely, however, that certain functional murmurs are due to changes in the vascular walls, owing to the poor nutrition, due to anemia. We may readily conceive that the aorta or pulmonary artery might under such conditions change sufficiently in calibre relatively to the firmer ring at the base

as to permit the formation of eddies and consequently of murmurs. The well-recognized yielding of the mitral ring in the poorly nourished offers a sufficiently close analogy to render this explanation of certain hemic murmurs a probable one.

Occasionally a so-called functional diastolic murmur is undoubtedly due to definite regurgitation from either the aortic or pulmonary orifice. The inconstant character of the murmur and absence of post mortem evidence of valvular disease point to a yielding of the ring supporting the valve as the real cause.

Certain murmurs doubtless originate in the cervical veins, since they may be stopped by pressure upon them. A further discussion of the origin of functional murmurs would involve so much of theory and conjecture as to be out of place here.

Functional murmurs are less constant than organic ones, more likely to be systolic in time, are not commonly associated with enlargement of the heart nor with signs of failing circulation; are as a rule softer in character, and much less likely to be associated with thrill; are not usually transmitted according to the usual rules known to apply in the case of organic murmurs; are more influenced by respiration and are much more likely to be associated with anemia.

*Cardiorespiratory Murmurs.*—A systolic murmur is often to be heard at the apex, commonly regarded as being due to the forcing out of the air contained in the vesicles and bronchioles of the lappet of the lung overlying the apex, by the compression exerted by the heart in systole. It is best heard during systole, along the left side of the heart and especially during a long slow inspiration, when air is actually entering the portion of lung involved. The murmur is not transmitted in the direction of that of mitral regurgitation. A burst of fine moist rales may be heard if secretion be present in the alveoli or tubes. Pressure with the stethoscope bell or the ear may make them more distinct.

This cardiorespiratory murmur is of importance chiefly as a cause of confusion to those not expert in physical examination. Many applicants for insurance have been referred to me after having been unjustly credited with having mitral disease, by the medical examiner, and doubtless many men are refused a certificate of health

through the same error. If the phenomenon has any pathological significance it is probably nothing more than that slight pleural adhesions prevent the normal motility of the lung at the place involved.

*Arterial Murmurs.*—These are systolic in time unless due to aortic regurgitation, when they may be diastolic. Pressure upon an accessible artery with the bell of the stethoscope produces a systolic murmur by interfering with the calibre of the vessel and the normal passage of the blood stream. If the vessel be narrowed or distorted, or its interior surface be roughened by atheroma, a systolic murmur and even thrill may be noted. Over the anterior fontanelle in babies, and over the pregnant uterus the murmur is present, but not explicable by reference to any of the causes mentioned, the arteries being presumably normal. The murmur noted so frequently over the subclavian artery in tuberculous patients probably owes its origin in part to compression or distortion due to pleural adhesions and in part to pressure by the stethoscope. That over the aorta arises from atheromatous roughening of the intima of the vessel, or is transmitted from a diseased aortic valve. Hardening of the arteries elsewhere is commonly present. The heart sounds are transmitted to some extent into peripheral vessels. In aortic regurgitation, apparently owing to the sudden distention of the arterial walls, the systolic wave produces the intense “pistol shot” sound. Duroziez’s diastolic sound may also be present.

*Venous Murmurs.*—Venous hum in the neck, noted in anemia, may assume a diastolic or systolic character, audible over the base of the heart in rare cases. The murmur may be abolished in many cases by the assumption of the recumbent posture, or stopping of the venous flow by pressure.

The sound of closure of the valves in the jugular vein has been heard in tricuspid insufficiency, but is interesting rather than clinically important.

*ORGANIC MURMURS.*—These are produced by eddies in the blood stream resulting from changes in the structure or relationship of the different portions of the heart and the vessels adjacent. The valve may be in part destroyed, as by endocarditis, so that it cannot effectually stop its proper orifice (absolute insufficiency); or the ori-



fice may have become so stretched that the normal valve cannot stop it (relative insufficiency)—murmurs of regurgitation. It may have become adherent at its edges or narrowed by cicatricial contraction, so that the blood current is obstructed—murmur of stenosis.

A systolic murmur may be produced by such roughening of the basic valve or the aorta or pulmonary artery in relation with it as suffices to produce eddies in the blood stream, although no material obstruction be present. A dilatation of either of the vessels mentioned may cause so great a relative difference in size as compared with the valvular orifice adjacent as to produce a systolic murmur similar to that of stenosis.

Stenotic murmurs are also spoken of as obstructive and those of insufficiency or incompetency as regurgitant. Systolic murmurs originating at the basic valves may indicate obstruction, and at the auriculoventricular orifices may point to regurgitation, while diastolic murmurs originating at the base are presumptive evidence of insufficiency, but point to stenosis if heard at the auriculoventricular orifices. In a general way we may assume that the left-sided murmurs are frequent and more often acquired, while right-sided ones are infrequent and generally congenital. Under the term diastolic, as applied to heart murmurs, we include as a sub-variety the pre-systolic murmur generally associated with stenosis of either one of the auriculoventricular valves.

Firstly, of prime importance in studying a cardiac murmur is the determination of its *time or place in the cardiac cycle*. This is most easily decided by reference to the visible, audible, or palpable apex-beat or the carotid pulsation, which is for practical purposes coincident with it. The systolic murmur begins with the systole of the heart in most cases, but the murmur of mitral regurgitation not infrequently is late systolic in time. In the diseased heart the first and second sounds may so nearly resemble each other that care should be used to determine absolutely the time of a given murmur by fixing the time of systole as above indicated. Since a murmur may replace a normal cardiac sound the need of the utmost care is evident. Diastolic murmurs may begin with the closure of the basic valves and persist until the beginning of systole, as is typically the case in

leakage at the valves mentioned, or may, as is more commonly the case when obstruction exists at one of the auriculoventricular valves, begin or at least first become audible somewhat later than the time of the second sound and run into the coming systole—presystolic murmur. A mid-diastolic murmur is possible and probably indicates the same obstructive lesion as the presystolic murmur mentioned.

Secondly, we should find the *place of maximum intensity* of the murmur. If this be at the "aortic area" we assume that the aortic valve is the seat of the murmur, although the murmur of regurgitation is best heard along a line running from this region toward the apex. If to the left of the sternum in the second interspace we assume that the murmur arises from the pulmonary valve. Mitral murmurs are best heard as a rule close to the point of the apex-beat, but may be internal to it or along a line passing upward to the pulmonary area. Tricuspid murmurs have their areas of maximum intensity at or near the root of the ensiform cartilage, upon either side of the median line.

Thirdly, we study the *area of diffusion* of the murmur, for it is likely to be propagated in some particular direction, the consideration of which will determine its point of origin if this be in doubt. In general we may trace the murmur, noting a progressive diminution in its intensity, to its point of complete disappearance, but three exceptions should be noted. The mitral regurgitant murmur may be louder in the back than along the line leading under the left axilla to this point; the murmur of pulmonic obstruction may be lost as we follow it upward from the pulmonic area, but yet be found in almost its full intensity in the upper interscapular space; the third exception pertains to the diminution in intensity of the aortic regurgitant murmur in its course under the right ventricle, and its reappearance, perhaps changed in quality, at the apex.

If more than one murmur be present, by tracing one toward the valve from which the other murmur originates we may often find a point where its characteristics begin to be overshadowed by those of the other murmur. The increase in intensity after passing this point suffices to establish presumptively the presence of the second murmur, and its character and origin are made out by following

the usual rules. Fortunately two systolic murmurs at the apical region, for instance, are rarely of the same pitch or quality, these points of difference giving material aid in differential diagnosis. Circulatory failure, evidence of dilatation or hypertrophy of certain cavities, change in other cardiac sounds, e. g., pulmonary accentuation in mitral disease, changes in character of the pulse, etc., should be sought for as confirmatory evidence in every case.

Fourthly, we investigate the *quality* of the murmur. The most frequently heard is the blowing sound fairly characteristic of regurgitation at either of the four orifices. The long soft distant blowing of regurgitation at the basic valves may be distinguished, wholly aside from the time of its occurrence, from the shorter, sharper blowing of mitral and tricuspid regurgitation, and the latter further tend to diminish toward the end in a fairly characteristic way.

Then we have the harsh, rough murmur, especially characteristic of obstruction at the basic valves, of the same duration as the systolic apical murmurs, and generally indicating a roughened orifice. The harshness is more marked than in the harsh variety of auriculoventricular systolic murmur.

Most certainly diagnostic of all is the blubbery murmur of mitral and more rarely of tricuspid stenosis, the "presystolic roll." Its quality alone suffices for the diagnosis of auriculoventricular stenosis if we except the rare Flint's murmur. (See page 266.)

Musical murmurs have a definite musical pitch, but are of no especial significance on account of this quality, excepting that they are generally organic. They are nearly always systolic, generally mitral in origin, and practically always mean absolute insufficiency of the valve.

The harshest murmurs are heard at the aortic area and generally signify calcareous roughening and narrowing of that orifice. The rougher the quality of any murmur the more likelihood there is of finding a palpable thrill. On the other hand, thrill is most common with the blubbery presystolic murmur of mitral stenosis.

The auriculoventricular murmurs indicative of stenosis are the only ones which increase in intensity to the end.

Fifthly, the *intensity* of the murmur demands attention. It may

be so great that the patient is conscious of hearing the abnormal sound and the physician may hear it at a distance of several feet. It is unfortunate that certain physicians regulate the gravity of their prognosis and the size of the dose of digitalis by the loudness of the heart murmur, when, as a matter of fact a loud murmur is often an indication of an excellent state of compensation. The failure in intensity, even to disappearance, of mitral murmurs in particular, with a break in compensation, is well recognized, and the reappearance of the murmur may be the first sign of improvement. On the other hand, the gradual increase in intensity of a systolic murmur at the apex in a case of acute rheumatism is to be taken as a sign of increasing valvular damage.

The intensity of a cardiac murmur depends upon the force and speed with which the blood current is driven over the diseased valve and is therefore greatest, in a general way, in systolic murmurs of the left heart, and least in diastolic murmurs of the right heart, the systolic murmur of the right side and the diastolic murmur of the left being intermediate in order. The presystolic murmur of mitral stenosis is much louder than the corresponding one upon the right side.

The development of anemia often leads to a harsher quality in a given heart murmur. Obstructive murmurs at the mitral and tricuspid orifices are loudest when the patient is in the upright posture, while the corresponding regurgitant murmurs are loudest when the subject lies down. The basic murmurs are less influenced by change of posture.

Sixthly, the *length* of the murmur is of interest. The longest ones are those of aortic and pulmonary insufficiency, filling the whole diastole. The shortest are commonly those of regurgitation at the auriculoventricular valves, or obstruction at the basic valves, these filling the whole systole in most cases. The murmur of stenosis at the same valves may fill the whole diastolic pause, but more commonly occupies only the latter portion of the interval, being, in other words, presystolic. No deductions can be drawn as to the severity of the disease from the length of the murmur, excepting as it is taken in connection with all the other factors in the examination.

*Other Features.*—Apart from the rapid development of a hemic murmur, after a severe hemorrhage, the sudden appearance of a murmur, commonly after severe exertion or during the progress of a septic or other severe endocarditis, signifies a sudden change in the structure or supports of the valves. I have twice confirmed the suspected rupture of an aortic cusp by post mortem examination, the diagnosis being based upon the known endocarditis and the instant development of the signs of fully developed aortic regurgitation. A change in the character of the murmur may be accounted for by breaking away of the vegetation from the edge of the valve or rupture of one of the chordae tendineae. The latter event may occur after severe exertion apart from the acute disease. Exercise may change the character of the murmur by raising the blood pressure and making it more distinct, by tiring the heart muscle and making the murmur less distinct, or, in the latter event, may develop a murmur by permitting the relaxation of exhaustion to occur at the auriculoventricular ring, with the production of regurgitation not present before.

Deep inspiration renders many organic murmurs less easily perceptible through overlapping of the heart by the distended lung. The cardiorespiratory murmur above mentioned is better heard under these conditions.

**Relationship Between Murmur and Normal Cardiac Sounds.**—Murmurs at the different valves may completely displace or cover up the normal sound there produced, and in general are of graver significance the more this feature is in evidence. If an aortic regurgitant murmur apparently fills the whole diastolic interval and yet the valve sound is distinct, we may infer that at least one of the valves is fairly serviceable. A late systolic murmur at the mitral area probably signifies comparatively slight leakage, the valves holding for an instant, or else asynchronous contraction of the two ventricles,—the first sound of the heart being produced by the right ventricle, and the murmur by the defective mitral valve. There seems to be no especial relationship between the time of appearance of the presystolic murmur in the diastolic pause and the severity of the obstructive lesion.

**CHARACTERISTICS OF THE SPECIAL VALVULAR LESIONS.**—For the diagnosis of a valvular lesion we must have certain signs and symptoms, and we shall here try to put together those features upon which we base the diagnosis of an individual lesion.

As we have obstruction, stenosis, or leakage, regurgitation, at either one of the four valves of the heart, we must study the characteristics of the four obstructive murmurs and of four regurgitative murmurs. The obstructive murmur is due to definite organic narrowing of the orifice in question, either congenital, or brought about by the presence of vegetations in acute endocarditis, or as the result of the slow cicatricial contraction and thickening, often with the deposit of lime salts, which follow an endocarditis. All of these steps may be noted in the lesions producing leakage, but in addition there may be leakage from rupture of a valve, of its supporting chordae tendineae or the papillary muscle, or because of the stretching of the fibrous ring supporting the valves—relative insufficiency. These last mentioned factors account in part for the fact that regurgitation is so much more frequent than obstruction when we consider the sum total of valvular diseases. It should be noted that both obstruction and leakage may be present at the same time in the same valve. If the edges of the valves be partially grown together by adhesive inflammation, and at the same time the free edges of the valves be damaged by endocarditis, we have obstruction because the valves cannot open to the full extent, and regurgitation because their edges are so damaged that they cannot close perfectly in their free extent. Most obstructed valves also leak, because the obstruction signifies actual organic change in the valve. The converse does not hold true, since most regurgitations are due to functional trouble with the muscles supporting the valves and not to the changes of endocarditis.

*Aortic Obstruction.*—Actual obstruction at the aortic orifice of sufficient degree to be capable of diagnosis is the rarest of left-sided diseases. The murmur should be: (1) in time,—systolic or late systolic; (2) place of maximum intensity,—right of sternum at aortic cartilage; (3) area of diffusion,—upward into carotids and to some extent into the vessels with the blood current; (4) quality,—

generally rough and harsh, occasionally musical; (5) intensity,—great; (6) length,—that of the systole of the heart. Systolic thrill often accompanies the murmur and the diagnosis is much safer if the sign be present. It may be exceedingly harsh and transmitted into the vessels. The pulse is likely to be infrequent, small in height, and firm to the touch. Because of the obstruction, moderate hypertrophy of the left ventricle is commonly present, and the apex-beat is slow, forceful and deliberate. The second sound is often absent or feeble owing to the structural damage to the valves, and their consequent inability to close sharply. The obstruction is organic in character.

*Aortic Regurgitation.*—Aortic regurgitation, which is more common than stenosis of this valve, gives rise to a murmur which is: (1) in time,—throughout the entire diastolic period typically, often not heard in the latter part; (2) place of maximum intensity,—downward and inward from aortic cartilage, not infrequently at the apex; (3) area of diffusion,—toward apex, rarely outside the precordial area; (4) quality,—soft and blowing, occasionally musical, often seeming distant and faint; (5) intensity,—moderate, often faint; (6) length,—full period of diastole, or faint, even disappearing, toward the end.

The heart area is much enlarged in well-marked cases, downward and to the left; systole of heart, powerful and “lifting”; aortic sound often absent; Corrigan’s pulse, capillary pulse, “pistol shot” (systolic), and Duroziez’s signs (diastolic) in the vessels. Diastolic thrill is common. The faint murmur may be better appreciated with the naked ear or the phonendoscope than with the stethoscope. As compensation fails it may disappear. The murmur is one of the most reliable in diagnosis of heart murmurs. The leakage may be due not only to organic change in the valve but to rupture of the cusps or stretching of the ring.

*Mitral Obstruction.*—The murmur should be: (1) in time,—presystolic, may fill the entire diastole; (2) place of maximum intensity,—small region around the apex-beat; (3) area of diffusion,—but little beyond this; (4) quality,—rumbling, purring, vibratory, ending with a sound like r-r-R-R-P, at the occurrence of the first

sound of the heart; (5) intensity,—marked, may disappear with loss of compensation and reappear later; (6) length,—all or any portion of the time between closure of the basic valves and the first sound of the heart, perhaps most commonly the last half or two-thirds of the period mentioned.

Moderate hypertrophy of the heart, especially of the left auricle and right ventricle, with consequent transverse enlargement, a small pulse, frequently irregularity of the heart beat, and absence of second sound of the heart at the apex may be noted. The basic sounds are often doubled or “split.” Presystolic thrill is often present. Stenosis is due to organic valvular disease.

*Mitral Regurgitation.*—The murmur should be: (1) in time,—systolic, often late systolic; (2) place of maximum intensity,—at or near apex, especially slightly outside of it; (3) area of diffusion,—typically to the left to axilla, often to back, occasionally upward toward pulmonary area; (4) quality,—soft, blowing, musical; (5) intensity,—moderate, but often loud, frequently in broken compensation so feeble as to become inaudible; (6) length,—full time of systole, or in late systolic murmurs, the latter portion of systole; generally tapers off at the end.

With the murmur we have enlargement of the heart transversely, accentuation of the pulmonic second sound, not infrequently systolic thrill; irregularity of the pulse, frequently extreme, is a common feature late in the disease.

The murmur may be due to, and in perhaps half the cases is due to, relative insufficiency of the valve, and not to organic disease.

*Pulmonic Stenosis.*—The murmur should be: (1) in time,—systolic; (2) place of maximum intensity,—to the left of the sternum at second space; (3) area of diffusion,—slightly around this point and to the back between scapulæ, following the general direction of the pulmonary artery; (4) quality,—generally harsh; (5) intensity,—loud; (6) length,—full period of systole.

The lesion is rare, likely to be due to congenital malformation, often accompanied by systolic thrill, and the pulmonic second sound is often absent. Always organic in character. Difficult to exclude



hemic murmurs and congenital disease of nearby portions of the heart.

*Pulmonic Regurgitation.*—The murmur of pulmonic regurgitation should be: (1) in time,—diastolic; (2) maximal intensity,—at and below the pulmonic area; (3) area of diffusion,—downward toward tip of sternum, not to apex nor to neck; (4) quality,—soft; (5) intensity,—feeble; (6) length,—full time of diastole.

The murmur is rare excepting when due to congenital deformity of the valve, when thrill may be present, or when due to temporary overstretching of the pulmonary ring in mitral stenosis and fibroid phthisis. (After overtraining in one instance). There is no Corrigan's pulse nor capillary pulse; no marked enlargement of the heart nor other signs of aortic regurgitation. Pulmonic second sound is feeble or absent. It may be organic or relative.

*Tricuspid Stenosis.*—Tricuspid stenosis should give murmur: (1) in time,—presystolic, may fill entire diastole; (2) maximal intensity,—the tricuspid area; (3) area of diffusion,—not outside this area; (4) quality,—same as that of mitral stenosis but less sharply defined; (5) intensity,—less than that of mitral stenosis, which generally accompanies it; (6) length,—same as in mitral stenosis.

This is always organic and 97 per cent. of such cases are associated with mitral stenosis. There is the same type of murmur, and same enlargement of heart. Different area of greatest intensity and possibility of thrill separate from that of mitral stenosis or murmurs of different quality, or both, offer possible grounds for differential diagnosis during life. Presystolic thrill may be present.

*Tricuspid Regurgitation.*—Tricuspid regurgitation should give a murmur: (1) in time,—systolic; (2) maximum intensity,—tricuspid region, fifth left cartilage and to right; (3) area of diffusion,—not far from this point; (4) quality,—soft blowing; (5) intensity,—feeble; (6) length,—that of systole.

Area of heart is increased slightly transversely (region of right auricle especially). Thrill may be present; venous pulse, in neck and in liver; and cyanosis. It is commonly associated with mitral regurgitation, but the murmur may be of different quality from the

latter. It may be organic, but is vastly more common as relative leakage.

COMBINED VALVULAR LESIONS.—It is very common to have lesions of more than one valve present and still more common to have several murmurs present. As to the first proposition, it is not at all strange when we consider that the causes of valvular disease are general ones, rheumatic or septic endocarditis and atheromatous changes, which may well operate upon several different valves. As to the second, we should consider that a serious change in one valve may easily disturb the circulation not only through the cavity affected most directly, but even in cavities of the opposite side of the heart indirectly. Thus a well marked mitral stenosis may give a presystolic murmur when compensation is good. When it begins to fail a relative mitral regurgitation is often heard with it. As further failure occurs, we may find the veins of the neck pulsating and hear the soft systolic murmur of tricuspid regurgitation near the tip of the sternum; in the effort at compensation the pressure in the pulmonary artery may be so raised by the hypertrophied right ventricle that we finally may hear the soft distant diastolic murmur beneath the pulmonary valves which signifies that they have yielded and permit leakage. But one organic lesion is present, yet three valves have given way under the strain of increased pressure in the efforts at compensation. The presystolic murmur at the apex in aortic regurgitation, Flint's murmur, has not even so well defined a basis as that in the case of relative insufficiency at the mitral valve, and the basic systolic murmurs are so often due to roughening of the orifice upon the left side of the heart and to hemic causes upon the right that we must not at all assume that a murmur necessarily indicates demonstrable valvular disease. We should endeavor to explain all the trouble we find in the heart upon the basis of the actually indisputable lesions we find, bearing in mind that such organic disease as an explanation of the secondary murmurs is easily possible. Thus the presystolic mitral murmur is presumably of the Flint type in case of well marked aortic regurgitation, yet I have twice demonstrated the correctness of my diagnosis of aortic insufficiency and mitral stenosis in conjunction, at the post mortem, and

have made the diagnosis with equal confidence in a third case still living.

The Flint murmur, almost indistinguishable from that of mitral stenosis, is probably dependent upon the changes in the relationship between the mitral valve curtains and the size and shape of the left ventricle, as the latter is modified by the dilatation due to the aortic insufficiency. It is probable that eddies in the blood current through the mitral valve and in the ventricle, set up by the leakage from the aortic valve, form the basis of this presystolic murmur.

Combined lesions in a single valve are present more frequently than our records would indicate. It is well known that most obstructive lesions so damage the valve leaflets that sufficiently exact apposition to prevent leakage is not probable, yet the murmur of regurgitation is often not demonstrable during life.

The most frequent combinations in a series of 600 private cases of heart disease were as follows: mitral regurgitation with mitral stenosis, in 44 cases; mitral regurgitation and aortic regurgitation, in 31 cases; mitral regurgitation and tricuspid regurgitation in 8 cases; mitral stenosis with associated relative pulmonic regurgitation in 3 cases; various other combinations of lesions were noted less frequently.<sup>1</sup>

The diagnosis of the double mitral lesion may often be made by the demonstration of increased cardiac breadth, accentuation of the pulmonic second sound, signs and symptoms of respiratory and circulatory difficulties, and of systolic and presystolic apical murmurs. Yet either murmur may predominate in different stages of compensation, and either may disappear. The murmur of leakage when due to structural damage of the mitral valve, as we assume in this case, is very much more constant than that due to the stenosis, for the latter may reappear and disappear several times in a year under conditions of improvement, and failure in cardiac strength. The advent of stenotic changes, as a result of the continuance of the contracting cicatricial process in the valve, often leads to some improvement in the patient's condition by correcting to some extent the amount of the leakage.

<sup>1</sup> Reported in *Medical Record*, Nov. 3, 1909.

The history of repeated pulmonary hemorrhages, so much more common in stenosis than in regurgitation, and the vigor and sharpness of the first sound at the apex, may lead us to suspect stenosis, even in the absence of a presystolic murmur and thrill.

*Double Aortic Disease.*—A double murmur at the aortic orifice is often significant of obstruction and leakage combined. We should note, however, that the regurgitative murmur is one of the most dependable in diagnosis and that the direct murmur is far more often due to roughening of the orifice or atheroma of the beginning of the arch than to any material obstruction. In the absence of systolic thrill, we should be chary of making a diagnosis of the obstructive lesion, and attribute most of the symptoms to the leakage.

*Aortic Regurgitation and Mitral Disease.*—Both the valves under consideration are likely to be affected in acute rheumatism and other infectious diseases, and consequently organic disease is common in both regions at once. Yet relative leakage at the mitral is so frequent as the left heart dilates under the strain of the regurgitation from the aortic valve that we should give such a possibility the first consideration. The signs of aortic disease are less sharp if the mitral lesion be present—capillary pulsation, Corrigan's pulse—and the presence of the mitral disease adds greater transverse breadth to the cardiac area than we commonly find in pure aortic regurgitation. Dyspnea, accentuation of the pulmonic second sound, and edema or other signs of circulatory failure are more likely to be present.

*Mitral Stenosis and Tricuspid Stenosis.*—It has been mentioned that the latter disease is rarely present, excepting in combination with the former, both being due to the same general endocarditic process. The presence of two presystolic murmurs at the apex, generally differing somewhat in character, and especially the presence of two distinct thrills, may lead to the correct diagnosis.

The murmurs of relative insufficiency at the tricuspid and pulmonary valves under conditions of increased stress, are so much more frequent than organic trouble at these locations that we should beware of attributing the murmurs to structural valvular changes, no matter what the character of the organic valvular disease with which they may be found.

Constancy of the murmur and steady increase in symptoms attributable to the insufficiency, give ground for the suspicion that the changes are of organic nature.

## THE LUNGS

### A. PERCUSSION OF THE LUNGS

Over the areas which are underlaid by normal lung tissue we have vesicular resonance, dependent upon the presence of air in the alveoli of the lung. The normal resonance here is low in pitch, of long duration, of great intensity, and varies slightly, according to the area percussed, the thickness and elasticity of the chest walls, and the size and nearness to the surface of the bronchi. The part most deficient in vesicular resonance in the normal chest is the right apex, owing to its cone-shaped structure, the lesser proportion of vesicular tissue here as compared with the space occupied by the bronchial tubes, and the proximity of the trachea.

If the air vesicles be filled with exudate in a limited area, as in bronchopneumonia, with normal lung tissue around the part affected, we shall have a slight impairment of resonance or slight dulness with a sound higher, of less duration, and of less intensity. If a considerable area be involved, as in lobar pneumonia, with complete filling of the alveoli and smaller bronchi, flatness is produced with intensification of the changes from normal vesicular resonance already mentioned. The height of flatness with lung tissue still in contact with the chest wall is reached in massive pneumonia in which all alveoli and bronchi are filled solidly with fibrinous exudate. A similar condition of absolute flatness is brought about by solid tumor of lung, or vastly oftener by accumulation of fluid within the pleural cavity, crowding the lung away from the chest wall. Flatness to moderately strong percussion is to be found over the triangle of absolute cardiac dulness and over the portions of the liver in contact with the chest wall. In both cases relative dulness is present at those areas where a shelving edge of lung projects over the retreating portion of the solid organ beneath. The flatness of the heart under the sternum has a peculiar bony resonance, and is less perfect than that over the

upper sternum when a large aneurism or solid tumor displaces the normal lung tissue of this region.

**Normal Outlines of the Lung.**—By percussion we may determine the borders of the lung, and we find that they vary greatly in position in health and disease with changes of depth of respiration, size of the heart, condition of the abdominal organs, etc. The tip of the lung normally rises an inch and one-half above the clavicle, modified by the depth of respiration, by any adhesions present (tuberculosis), by distension from emphysema, the conformity of the thorax, etc. On the right side below, the border is normally at the fifth space in the nipple line, crosses the sixth rib in the axillary line, and reaches the top of the eleventh rib at the spine. Upon deep inspiration the lung is pushed down one or two inches or more into the pleural complementary space. The mobility of this lung border of resonance is important in judging as to the presence of pleural adhesions here, especially when surgical intervention in this region is being considered.

The lung borders in front follow the lines of absolute cardiac dulness already described and are much modified by the presence of emphysema, lateral displacement of the heart, pleural and pleuro-pericardial adhesions, aneurism, tumor, etc. One of the signs of adherent pericardium is the immobility of the lung border of resonance upon inspiration and expiration, since pleural adhesions are often present as a result of extension from the pericardial inflammation.

Upon the left side the stomatic tympany interferes with the percussion of the lung border in front. In the axillary region and back the left lung is commonly found a little lower than the right, since the liver slightly interferes with the descent of the diaphragm upon that side.

The lung is probably affected by irritants applied to the outside of the chest wall. Abrams applies the term "lung reflex" to the supposed local and temporary dilatation of air cells believed to result from applications of cold, etc. Cabot believes that a similar condition results from percussion if long continued. I know of no practical application of what knowledge we possess in this field.

In the chest we find tympany normally over the left lower ribs in the region occupied by the cardiac end of the stomach, and varying greatly, according to the amount of gas present. The surrounding liver, lung, and spleen delimit it on the right, above and to the left, and the tympanitic note runs into a more or less similar one, proceeding from the more or less distended gut below. No area of the body presents so many variations as to percussion values as this one, and in no area are mistakes more frequent. The possible presence of liquid contents in the stomach, of air in the pleural cavity (pneumothorax), of subphrenic abscess with gas and movable dulness, or of diaphragmatic hernia, may all demand consideration. In ascites and abdominal tumor the upward displacement of the stomach may add to the confusion.

Tympanitic resonance over the lung area may also occur because of the presence of air or gas free in the pleura (pneumothorax), or in a large cavity, or because the tension of the lung is relaxed, and it thus loses its vesicular qualities of resonance upon percussion, as in pneumonia and pleuritic effusion (skodaic resonance).

If the lung be over-distended, as in emphysema, we have the exaggerated vesicular resonance which characterizes the condition, the pitch being higher if the tension is high, as in acute emphysematous conditions, lower if it be lowered, as in chronic senile emphysema.

Over-filling of the lungs by a deep breath, or especially by rapid deep breathing, as after short, severe exertion, changes the character of the resonance appreciably, giving a slightly "emphysematous" tone to the note. A small area of consolidation in one apex may thus be outlined by contrast more easily with the lungs distended.

By the cavernous percussion note we mean the variety heard over pulmonary cavities lying near enough to the surface and of sufficient size to be accessible to successful investigation by this method. The note varies greatly, according to variations in the two conditions mentioned and according to the shape of the cavity, its condition of fullness or emptiness, as to whether or not it connects with a bronchus, and the condition of the surrounding lung tissue. The note varies much at different examinations because of the variability of several of the above conditions. It is impossible to describe in words the

variations mentioned, though they are not difficult to appreciate upon demonstration. Typical cavernous resonance is found when the tension in the cavity is low. The amphoric resonance is found with closed cavity with more rigid walls.

The most characteristic sound heard in connection with pulmonary cavity is the so called "cracked pot" sound. It is best heard when a cavity connects with an open bronchus and lies near to the surface of the lung, especially in the upper lobe. Percussion should be forcible, the mouth should be open, and if possible the bell of the stethoscope should be held in front of the patient's open mouth. A similar sound may be produced in children with very elastic chest walls by forcible percussion, but it is not limited to any one area, as is the case with that from a cavity.

Less attention is paid than formerly to the Wintrich phenomenon,—that rise in pitch upon opening the mouth while percussing over a cavity; to Friedreich's change of note while percussing during forcible inspiration and expiration, varying the amount of air and the tension in the cavity; and to Gerhardt's sign,—the change in the note with change in the posture of the patient. It is probably due to change in the shape of the air-containing portion of the cavity by movement of the contained fluid.

Many cavities are so situated as to be easily recognized by the ordinary signs, while many smaller ones, remote from the chest wall, are first found post mortem. Variations in conditions of fullness of cavities, and of patency of the communicating bronchi, render it necessary that several examinations be made, preferably at different times of the day, before passing definitely upon their presence or absence. Most of those which for various reasons I have had drained by the surgeon, have proved to be smaller than they were believed to be.

## B. AUSCULTATION OF THE LUNGS

It is best to show the patient how he is to breathe before beginning the examination, or to tell him to breathe as he would after running up a flight of stairs. Noises made in the effort to breathe deeply are commonly corrected by the patient if attention be called to them.



Patients who fear that some lung trouble will be found and dread to know it sometimes wilfully refuse to breathe properly.

The normal breath sounds depend upon the movement of the air in the tracheobronchial system and the alveoli of the lung. One-third of the 30 cubic inches drawn in in quiet inspiration suffices to fill the trachea and bronchi, leaving the remainder to actually enter the aveoli. Much of the sound which we perceive in ausculting the chest arises from the vibrations set up as the air passes the chink of the glottis. The changes from normal quiet breathing relate either to variations in the rhythm, pitch, quality, intensity and duration of inspiration and expiration, or to adventitious sounds resulting from disease, such as the various rales, succussion, friction, etc.

We hear normally either vesicular breathing, over the alveoli of the lung, or the tracheal or bronchial type, best heard where the tubular air-conveying portions of the respiratory apparatus predominate, namely in the region of the trachea or primary bronchi. In children bronchial respiration is heard over more extensive areas because of the ready transmission of sound in the child's chest.

**Types of Respiration.**—**VESICULAR RESPIRATION.**—In normal breathing we hear over the healthy lung beyond the region of distribution of the large bronchial tubes, a “breezy” sound well compared to that produced by the escape of air, if we expire with the lips in proper position to give the sound of “F.” The pitch, although low, is slightly higher upon inspiration than on expiration, the intensity is greater and the duration longer. I believe the statement often made in works upon physical diagnosis, that the pitch of expiration is higher than that of inspiration, depends upon the fact that if a bronchial element be introduced the expiratory pitch is raised.

The vesicular respiration described is best heard over regions of the chest where large areas of alveolar tissue functionate actively, as in the axilla. All the characteristics of vesicular respiration are exaggerated if the respiratory movements be increased in activity, whether from exertion, or from increased function of one portion of a lung because of decreased function in another (compensatory emphysema). In children the exaggeration of the breath sounds is

termed "puerile respiration." The decreased respiratory sounds of emphysema and of senility give rise to the terms "emphysematous" and "senile" respiration.

The sounds produced at the larynx by the passage of the air current are transmitted by the current downward into the lung by the bronchi and are modified by the dampening effect of the alveolar tissue, and by the addition of sounds produced in the tubules and at the entrance of the bronchioles into the air vesicles. The expiratory murmur should naturally be feebler since the direction of the sound-carrying air current is reversed, and because no such active muscular element is present in expiration as we have in inspiration.

The typical vesicular sound is modified in different parts of the chest by the amount of alveolar tissue subjacent, the activity of the particular portion of the lung examined, and the nearness or remoteness of the larger bronchial tubes. In the right clavicular region the sound is more bronchial than upon the left, because of the size and peculiarity of the distribution of the branches of the bronchial tree present. Between the scapulæ a harsher, somewhat bronchial type of respiration is found, owing to the nearness of the primary bronchi and the lower end of the trachea. Over the thin borders of lung projecting downward into the pleural complementary space the vesicular sound is enfeebled because of the thinness of the layer of lung beneath, this being normally more marked on the right side in the lower back because of the upward protrusion of the liver and consequent diminution of space for lung tissue.

**BRONCHIAL BREATHING.**—Bronchial breathing is heard in health over the trachea and larger bronchi. The word "tubular" conveys the best idea of its character, the sound somewhat resembling that caused by the passage of an air current through a metal tube. The sound is high in pitch and harsh in character, these features being especially marked in expiration. This bronchial or tubular respiration originates in the sound produced at the larynx by the passing air current, this being transmitted by this current and by the tissues of the chest and respiratory apparatus. Variations noted in the character of bronchial respiration are easily accounted for if we consider the varying strength of the air current, the size of the opening of

the larynx, the position of the cords, and the condition of the mucous membranes.

The adult chest lends itself less readily to the propagation of the bronchial respiration than that of the child. We should therefore note that a type of harsh respiration normal over the back of a child of five years would be pathological in an adult. In a similar manner the finding in the right apex of harsh respiration is normal while the same sounds upon the left would indicate disease.

Typical tubular breathing may be heard over the consolidated area in acute pneumonia. If a layer of normal lung tissue intervene between the consolidation and the wall of the chest, the bronchial respiration is spoken of as *distant*. In massive pneumonia, in which the bronchial tubes are filled with fibrinous exudate, no bronchial respiration is to be heard. In children, on the other hand, exquisite bronchial respiration, originating in the compressed lung, may be found over a serous pleural exudate.

**BRONCHOVESICULAR BREATHING.**—By this we mean a mixture of the two types of respiratory sounds already described, varying in the proportions of the two ingredients. In passing from an area of pure bronchial respiration to one of pure vesicular respiration one may, at successive positions of the stethoscope, find every gradation from one extreme to the other. Between the scapulæ we may hear this variety of respiratory sound in health. The exaggerated respiratory sounds in the sound lung, when the opposite one is compressed by a pleural exudate, partake of the bronchovesicular type. None of the synonyms for this variety of respiration have displaced the term we have adopted above. Different gradations may be denoted by suitable adjectives, as when we speak of “markedly” or “slightly” bronchovesicular respiration.

**CAVERNOUS RESPIRATION.**—This term describes the hollow sound caused by the passage of the air current into or out of an empty cavity, most commonly one formed by destruction of lung tissue in tuberculosis, or by pulmonary abscess or gangrene, but occasionally the result of a pneumothorax connecting with a bronchus. It is necessary for the production of cavernous respiration that the cavity have sufficient flexibility in its walls to cause a change in its capacity in

the acts of respiration. The sounds are low in pitch and have the hollow sound familiar in blowing across the open mouth of a jar or bottle.

The high-pitched amphoric respiration has much the same significance as cavernous respiration, but is more often associated with pneumothorax.

**INTERRUPTED RESPIRATION.**—If the inspiration be “jerky” we speak of it as being interrupted, or cog-wheel respiration. It is not at all necessarily pathological, but is often seen in nervous individuals in good health, disappearing perhaps with rest or absence of nervousness or fatigue. It is here associated with irregular action of the respiratory muscles. The systolic type should be carefully noted. It is best heard during a long, slow inspiration, and over the lower left side of the chest in most cases. The interruptions in the respiratory rhythm may be easily determined to be synchronous with the cardiac systole. The resulting filling of the pulmonary vessels presumably forces air out of the air vesicles and finer tubules under certain conditions of disease. This type of interrupted respiration commonly indicates demonstrable disease of the lung or pleura over the portion of the chest in which it is found.

The irregular type of interrupted respiration is often associated with early pulmonary tuberculosis, and probably depends upon obstruction of finer bronchial tubes by catarrhal products. If constantly present it has a definite pathological significance.

The respiratory sounds often change suddenly, especially after a cough, due to dislodgement or movement of secretion from a bronchus, and the freer or less free entrance of air to certain parts of the lung. A similar change of very striking degree is occasionally seen, due to the shifting of a foreign body in a bronchus.

**ABSENT OR SUPPRESSED RESPIRATION.**—This is typically noted in massive pneumonia, no air entering the smaller tubules or vesicles of the affected area. An obstructed bronchus, pleural effusion or pneumothorax may give rise to complete suppression in a certain area. In the obese, feeble respiratory efforts may be inaudible owing to the thickness of the chest wall.

Variations in the respiratory murmur in the different portions of

the chest are produced by causes limiting or increasing the movements of the corresponding chest muscles. Thus a pleurisy may cause so much pain as to inhibit the action of the respiratory muscles with corresponding diminution of respiration and modification of the murmur upon the affected side, and increase of action and sounds upon the opposite side. The lung of the side upon which the patient lies when in the lateral position in bed shows much diminution in respiratory sounds. An enlarged liver or spleen, dilated heart, pneumothorax, pericardial or pleural effusion, paralysis of one side of the chest, etc., may produce marked modification of the respiratory sounds in different portions of the chest.

**Modifications Produced by Disease.**—(1) **VESICULAR RESPIRATION.**—The respiratory murmur is decreased by those agents or conditions which prevent free movement of air in the smaller tubes and alveoli. If limited to a certain area, the diminution may be due to the blocking of a large bronchus by a foreign body, by compression by an aneurismal or other tumor, enlarged glands, etc. If general the cause commonly lies in the trachea or larynx. Edema, spasm, diphtheritic membrane or pressure of a foreign body or an abscess may be found as the cause. Great dyspnea is a feature of importance.

The lessened power of inspiration and expiration in emphysema gives rise to a marked enfeeblement of the vesicular murmur. The shape of the chest and the history of the case make the explanation easy.

The presence of exudate in the bronchi, as in edema of the lung, bronchitis and bronchopneumonia, prevents the normal movement of the air current from which the vesicular murmur originates. The presence of pleural effusion, pneumothorax, tumor of the lung or pleura, aneurism, etc., may cause complete suppression. The lower right lung offers a diminished murmur in many cases of appendicitis since the action of the diaphragm is interfered with by pain. Pleuritic pain elsewhere may produce similar results. Paralytic affections of the respiratory muscles may produce unilateral or bilateral involvement of respiration, and this is often noted in marked senility.

Increase of the force of the vesicular murmur has been mentioned as a characteristic feature of the breathing in children; and in cases

in which the lung is compelled to do more work than normal, because of failure of function of the other one through disease, pressure, bronchial obstruction, etc.

(2) **BRONCHOVESICULAR RESPIRATION.**—The breathing becomes bronchovesicular in character over portions of the lung normally presenting a pure vesicular respiratory murmur, in those diseases bringing about some degree of solidification in the lung tissue. Such a change is especially frequent in the early stages of pulmonary tuberculosis. If a portion of the lung be compressed, or retracted because of its inherent elasticity, as for example, in pleural effusion, a similar change may be noted.

(3) **BRONCHIAL RESPIRATION.**—This is seen in parts of the lung where it does not occur normally, in those conditions bringing about solidification of the lung, or such compression as to abolish the function of the alveoli. It is most typically seen in croupous pneumonia, tuberculosis with extensive solidification of lung, and in pleural effusion in children. In adults a feeble tubular respiratory murmur is often found in pleural effusion. Anything producing solidification of lung tissue with remaining patency of the bronchi may produce this phenomenon. Any changes in the lung tissue, increasing the solid elements and decreasing the alveolar tissue, which lead to the production of bronchial respiration, tend to the production of a prolonged expiratory sound as well.

**Adventitious Sounds.**—In disease certain sounds are to be heard in the chest, dependent upon anatomical or functional changes in the alveoli, bronchi or pleuræ.

**RALES.**—These are sounds, commonly bubbling or squeaking, produced by the passage of the air current through bronchi of greater or lesser size, which are obstructed to some degree by liquid, commonly an exudate (serum, blood, mucus, pus), by other foreign matter, by spasm, by swelling of the mucous membrane, or by pressure from without.

**Moist Rales.**—If the tubes be obstructed by liquid exudate we have coarse, medium or fine moist rales, depending chiefly upon the size of the bronchi affected, but in part upon the character of the fluid contained. A fluid of some density (mucus, pus) in the largest

tubes gives very coarse rales, the extreme type being the "death rattle" heard when in the moribund or other unconscious state, fluid accumulates in the trachea and is moved by the inspired and expired air currents.

Coarse, harsh gurgling rales are common over the solidified lung of advanced tuberculosis, and especially if cavities be present. Medium and fine mucous rales are heard over regions in which medium and fine tubules are present if they are in part obstructed by fluid through which air can pass. If the bronchi be completely blocked as in massive pneumonia and atelectasis of lung, no rales are present.

Exquisite moist rales may be heard over the lungs of patients whose air vesicles and finer bronchi have been in part filled by fluid from without, as after partial drowning, or when an echinococcic cyst or empyema breaks into the lung, or the stomach tube, used in feeding the insane, enters the trachea instead of the esophagus.

Moist rales are most abundant toward the end of inspiration. In pulmonary tuberculosis, if none are heard in the suspected area, coughing often brings them out. The sharpness and "nearness" of moist rales are somewhat dependent upon the presence of more or less solidification of the surrounding lung tissue. If coarse "near" rales are found over an area devoid of larger bronchi we should seek for evidence of dilated bronchi or cavity formation.

*Dry Rales.*—These differ from moist rales by the absence of the definitely liquid or "bubbling" character. They presumably indicate a more "sticky" exudate than that which gives us the moist rale. The dry clicks found in incipient tuberculosis offer the best example of medium dry rales. The crepitant rales so abundant in early croupous pneumonia are much finer, while sub-crepitant rales are intermediate in character.

Typical fine dry rales may be heard upon inspiration in the lower back portion of the lungs when patients first assume the upright posture after being in bed some hours, and when, with the first deep breath, the finer bronchi are opened up by the entering air.

The first few deep breaths may give such crepitation, especially in the region below the axilla, in normal individuals, and the rales may persist, especially in those at or beyond middle age, more or less

indefinitely. Cough may bring them out abundantly for a short time. If dry crepitation persists after many deep breaths, a presumption of the existence of disease is established.

Although the claim has often been made that the dry rale is a pleural product, the preponderating evidence, in my opinion, is entirely on the side of its pulmonary origin. Thus at the time when fever, dyspnea, dulness and cough, with the knowledge we have of the disease, lead us to believe that the croupous exudate is forming in lobar pneumonia, the crepitant rale is heard. At this time the impression is that a multitude of individual crackles make up the general sound produced. As complete hepatization occurs they disappear, and with the first sign of softening of the exudate the crepitus redux appears, and, with the increase of softening and the appearance of expectoration, a gradual transformation occurs until moist rales replace the crepitation. The presence of crackling in the early stage of edema of the lung when no inflammatory changes are present in the pleura and the gradual development of finer and then coarser moist rales can hardly be explained by any theory which does not recognize the intrapulmonary origin of the earlier signs mentioned.

In favor of their pleural origin is the fact that the friction of the bell of the stethoscope upon the dry hair covering the chest produces a sound almost exactly like the crepitant rale of early pneumonia. By wetting or greasing the surface the phenomenon may be caused to disappear. It is well for the clinician to become sufficiently expert in using the unaided ear for auscultation to avoid the necessity of using the stethoscope under such circumstances, since the hair does not notably interfere with direct auscultation.

*Rhonchi: Musical, Sonorous and Sibilant Rales.*—These sounds are produced when the calibre of certain of the bronchial tubes is encroached upon by spasm, by the presence of thick, sticky exudate or foreign body within the tube, or by pressure from without, causing an interruption in the free passage of the air current, analogous to that produced by the reed of a wind instrument.

In asthma we have the greatest abundance and variety of rales, and here spasm of the bronchi and obstruction by tenacious mucus both contribute to their production. The rales are sonorous, squeak-



ing, piping, whistling, groaning, or hissing in character, depending upon the character of the obstruction and the size of the bronchi involved. Coarse moist rales may be present also. Cough or deep respiration may completely alter the picture by displacing the obstructing mass. A lessening of the spasm by the action of drugs or a decrease in the congestion of the mucosa as by adrenalin may bring about their disappearance in asthma. If the rales depend upon the narrowing of the calibre of a bronchus by pressure of an aneurism, exertion with a rise of blood pressure and greater distention of the sac may cause a decrease or disappearance of the sounds in accordance with the amount of pressure. If due to paralysis of one vocal cord, as by pressure upon the recurrent laryngeal nerve by enlarged glands or aneurism, the rales may be absent during quiet respiration, but very prominent upon violent breathing (as the "roaring" of horses due to glandular obstruction).

The hoarse, loud, sonorous rale produced by pressure upon, or internal obstruction of, the larynx, trachea, or primary bronchi is spoken of as "stridor." Tactile fremitus may be associated with the vibrations of this type of rale.

**FRICTION SOUNDS.**—Pleural friction becomes audible when the smooth surface of the pleural membrane becomes roughened by the loss of its normal moisture, as in cholera, or by the development of a fibrinous exudate, as in acute inflammations. The rubbing sounds are then heard in those portions of the chest where the respiratory excursion is great enough to permit sliding of the roughened pleural layers upon each other, hence most frequently in the lower lateral regions. Friction may, however, be heard anywhere or everywhere over the pleural distribution, and even below the lung margin in the pleural complementary space. Although predominating during inspiration, it is often heard during expiration, constituting a "to-and-fro" friction murmur.

In its incipiency, pleural friction may give rise to sounds resembling, if not identical, with, the so-called crepitant rales. If a liquid exudate is poured out the friction sound is softened, and a small amount of "slippery" purulent exudate may, as in the pericardium, abolish friction sounds and to some extent the pain associated with

the condition. Should a dry thick plastic exudate develop, the friction becomes loud and palpable. It may be grazing, rubbing, grating or leathery, like the creaking of dry leather. If a considerable pleural effusion be poured out, the layers of the pleura are separated below, and the friction at that point disappears, though still present above. The reappearance of friction at the upper limits of an effusion therefore indicates that it is subsiding.

In moderate breathing, the two inflamed layers of pleura may stick together, but the friction sound may be reproduced by deep breathing. It is more or less changeable in character from time to time, and we should not be too sure that a pleural friction has disappeared permanently since rest upon the affected side, followed by deep breathing after arising, unusual bodily movements, or especially placing the arm of the affected side above the patient's head, and directing that a deep breath be taken, may reproduce the lost sound.

In mild, chronic, so-called "rheumatic" involvements of the shoulder joint, and the fibrous tissues about the scapula, "shoulder creaking" may be present. It is produced entirely outside the chest cavity and may often be brought out during suspension of respiration by movements of the arm and shoulder. A misinterpretation of this phenomenon occasionally leads to a false diagnosis of pulmonary tuberculosis.

**OTHER ABNORMAL SOUNDS.**—There may be pathological sounds over the lung area not capable of assignment to any of the classes given above. They may be the "muscle sounds" already spoken of, or crumpling or crackling sounds of undetermined origin. After the healing of a dry pleurisy, in pulmonary tuberculosis, I have known a "crumpling" pleural sound, as I believe it to be, to persist for many years. These "indeterminate rales" probably signify the results of some pathological process, but we cannot always translate them.

**Metallic Tinkle.**—Over large cavities, occasionally in the lung, but more often in cases of hydropneumothorax or pyopneumothorax, we may hear just such a metallic tinkle as is produced by a drop of water falling to the surface of the water in a cistern. It appears probable that the explanation is rather the breaking of a bubble

of air admitted to such cavity from the submerged opening of a communicating bronchus. A definite bubbling sound may also be produced under those conditions.

*Succussion.*—If air or gas and fluid be present together in the pleural cavity the patient, by changing posture or by sudden bodily movement, may produce a splash audible to himself and to others. The physician may produce it by shaking the patient's chest, listening meanwhile with the ear applied to the suspected region. The succussion produced in the stomach by the sudden contraction of the diaphragm is easily distinguishable.

*Egophony.*—The "bleating" sound produced by the patient's voice, heard near the upper margin of a moderate sized pleural effusion, is of interest but entirely subsidiary in importance to the other signs considered. It is too uncertain to be of much value, and may even be present when solidified lung and not effusion is the cause of the dulness in the chest.

*Bronchophony.*—The humming sound of the voice over those portions of lung area devoid of large bronchi is spoken of as normal vocal resonance. Over the trachea and upper right bronchial region the voice is normally heard with great distinctness, but is muffled by the air-containing lung tissue elsewhere. As the air in the lung is displaced, as in pneumonia and tuberculosis, the voice sounds are more distinctly transmitted, provided the bronchial tubes remain open. If the tubes are prevented by any means—obstruction, or displacement from the chest wall by fluid effusion, thickened pleura, or pneumothorax—from transmitting the sounds propagated in them to the chest wall, decreased voice transmission results. In children it is not uncommon to hear the voice or cry through a serous effusion, bronchial respiration being heard well in these same cases.

Bronchophony is of much the same significance as increased tactile fremitus and is found commonly in association with it. It may be heard when the consolidation is sufficient for the production of tubular respiration. As in the case with the latter sign, bronchophony may be "distant," owing to the intervention between the area of lung in which it is produced and the ear of a layer of fluid, air

or alveolar tissue. Thus distant bronchophony becomes a valuable sign in the detection of a central pneumonia before percussion becomes available.

Pectoriloquy is an exaggeration of bronchophony, and signifies merely more perfect conditions for the transmission of the voice through the contents of the chest than are commonly present.

*Whispered Voice.*—The normal lung transmits the whispered voice as a very indistinct diffuse sound, especially at points distant from the trachea, and primary bronchi. If a moderate consolidation be present the sounds become more distinct, and such consolidation may generally be detected by means of the whispering voice before bronchophony is to be found. It is thus of value in localizing small patches of solidification as in bronchopneumonia. It offers the advantage of being less exhausting than the taking of the deep breaths or speaking aloud in feeble patients.

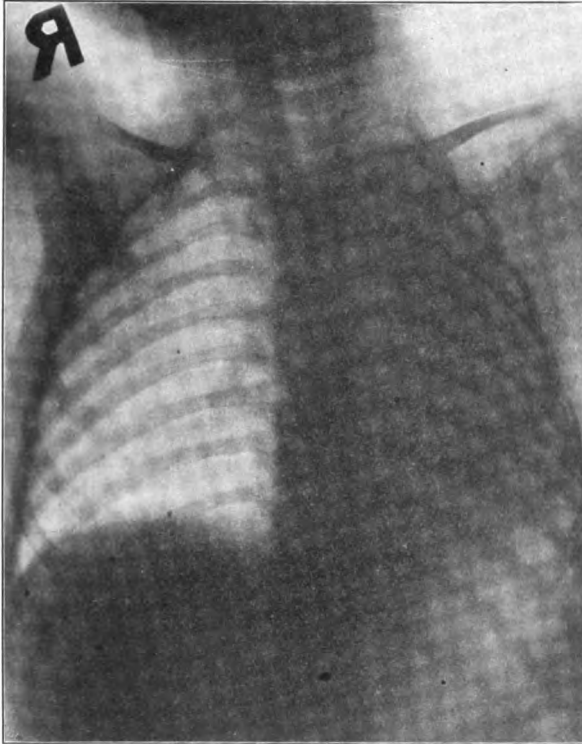
The transmission of the whispering voice through an effusion is of considerable value as evidence that the effusion is serous in character—Bacelli's sign. I have occasionally, however, heard the whisper exquisitely transmitted through a purulent exudate. A low pitched blowing whisper may be found over a phthisical cavity, while the solidified lung around it gives a whispering bronchophony. Flint terms the former the "cavernous whisper."

**Extraneous Sounds.**—Certain sounds originating elsewhere than within the chest may attract the attention of the examiner. We have already mentioned the muscle sounds. Creaking in the tissues of the shoulder joint as it moves slightly in respiration may closely simulate pleuritic friction, and I have known patients to be sent across the continent to a health resort for no more serious trouble, under the mistaken diagnosis of tuberculosis. The sound may frequently be produced by moving the arm while the breath is held. In surgical emphysema, as after fracture of ribs with perforation of pleura and lung, a crackling sound may be heard, somewhat resembling a crepitant rale, but evidently very superficial. Since the crackling is perceptible to the finger, we should not be deceived. After a hypodermic injection, and transfusion, when air has been admitted, in tissues infected with *B. aërogenes capsulatus*, and occa-

sionally after tracheotomy or after a severe paroxysm of whooping-cough, the same phenomenon may be noted. In the absence of air, the tissues distended by edema, or by fluid injected under the skin, may give a peculiar sound if the bell of the stethoscope be pressed firmly upon the skin. The crepitation produced by a broken rib or scapula may be heard.

**Foreign Bodies in the Air Passages.**—Pins, coins, and other small objects are often aspirated through the chink of the glottis and find their way into the trachea or bronchi. The larger ones are arrested at or above the bifurcation of the trachea, and fall more especially within the province of the laryngologist. Smaller ones are prone to fall into the larger right primary bronchus, not alone from its size, but from its more direct course and more powerful air current. If this bronchus be materially obstructed, respiration over the whole right lung becomes enfeebled, and vocal fremitus is diminished. Abram regards loss of vocal fremitus over an area still resonant, or even tympanitic, as one of the earliest and best signs of bronchial obstruction. Resonance is not at first impaired. If the obstructing body be drawn into one of the secondary bronchi the signs over the affected portion of the lung are likely to be more marked, and frequently moist rales and diminution of resonance are superadded as accumulation of the fluid takes place. In case the obstruction occur from a suppurating bronchial gland, as in one of my cases, these signs are extremely prominent, and an overwhelming bacillary infection may be noted in the sputum. The sudden change of location of the earlier signs, after a paroxysm of coughing, constitutes almost certain evidence of the presence of an unimpacted foreign body which has shifted its position. Fortunately most foreign bodies are of such a nature as to render detection by the X-ray easy and certain. Neurotic young women frequently claim falsely, and doubtless often believe, that they have just aspirated some object into the bronchi. In the absence at several examinations of any of the signs noted, and failing to find confirmatory X-ray evidence, I have found it fairly safe to dismiss such patients. One or two have afterwards confessed to the deception practiced.

After the unskillful use of the stomach tube in feeding insane, feeble, or hysterical patients, fearful dyspnea and cyanosis may be noted immediately after the food has entered the trachea. Abundant moist rales are present. If these patients survive the first onset of the suffocative symptom they may recover, as in one of my cases,



**FIG. 12.**—ANTEROPOSTERIOR VIEW OF THE CHEST OF A BOY 3 YEARS OLD, SHOWING A PNEUMONIA OF THE ENTIRE LEFT LUNG FOLLOWING THE INSPIRATION OF A BEAN WHICH LODGED IN THE LEFT COMMON BRONCHUS. (Dr. S. B. Childs.)

in which a pint of warm milk was fed into the trachea instead of the esophagus.

**Compression of the Trachea and Main Bronchi.**—Varying types of dyspnea are associated with the interference with the free passage of the air current, according to the cause. The elastic rings of the air tubes withstand pressure from without remarkably well, but gradually yield and permit a flattening of the tube if the pressure

continue, whether it be the steady inelastic pressure of a goiter or other solid tumor, or the intermitting pressure of an aneurism. After the rings lose their circular form their resistance to further pressure is but slight, and any sudden increase in pressure brings about instant and alarming dyspnea. Such sudden increase in pressure is more likely to occur in case of aortic aneurism or a very vascular thyroid growth than in malignant and glandular types of tumor. The temporary increase of blood pressure from the exertion even of undressing for examination brings about sufficient narrowing of the already collapsed trachea in aneurism to give, with the increase in the amount of air demanded by the exertion, signs of acute respiratory distress. The more sensitive the patient is in these respects the more the probability of aneurism or very vascular thyroid growth and the less of cancer or other solid tumor.

If dyspnea arise from change to a certain position in case of growth of any kind within the chest one may infer that the growth is so related to the air passages that the pressure becomes greater in the position of greater dyspnea. The type of dyspnea in case the left main bronchus is constantly somewhat compressed, and with exertion the right lung shows signs of deficient respiratory murmur, is commonly due to aneurism of the arch, which upon increase in its size from the exertion reaches further over and compresses the region of the bifurcation of the trachea and hence affects the right lung.

In dyspnea, paroxysmal or otherwise, we should not exclude the thyroid as a cause, because of inability to palpate it. The most dangerous cases are those in which the sub-sternal or "diving" thyroid is present.

## SECTION VIII

### EXAMINATION OF THE NERVOUS SYSTEM

The structure of the nervous system is so intricate and the functions are so complex that accurate diagnosis of the diseases which affect it is impossible without a thorough anatomical and physiological knowledge to start with, a careful consideration of the pathology, and finally, a method of examination more systematically arranged and followed than is used in the other departments of medicine. Fortunately the signs and symptoms point much more closely to the seat of the trouble and its nature than do those of the other systems, so that diagnosis becomes more intricate and more tedious, but more certain in the hands of experts, than in other departments. We have space only for a brief general review of the anatomical features involved.

### NEURAL ANATOMY

**The Neuron.**—The nervous processes of the body are carried on by the neurons, consisting each of a cell-body and processes extending out from it, the whole supported and bound together by the neuroglia.

A neuron consists of a single nucleated cell, in which the nervous impulse originates, and which possesses trophic functions, and its extensions by which nervous impulses are conveyed and received.

The dendrites are given off from one of the poles of the cell-body, and rapidly become more slender from repeated branching, finally terminating in delicate branches with slight thickenings at the end called gemules. Delicate neurofibrils pass from the dendrites into the cell and through it into other dendrites or the axon. The dendrites bring impulses to the cells from other sources, their functions being therefore cellulipetal.

The axon or axis-cylinder passes out from the body of the cell



and runs to a near or distant neuron, to muscle cells or secretory cells, according to its function. With these it communicates by means of its terminal arborization, the fibers interlacing with corresponding members of the other neuron. The axons may give off collaterals at right angles, which are either short branches immediately ending in end arborizations and communicating with nearby neurons in the gray matter or long branches reaching to more distant ones. The axons carry impulses away from the cell, being cellulifugal. The axon with its medullary sheath becomes a nerve fiber, and a bundle of such fibers constitutes a nerve trunk. We may assume that the neurons are morphologically distinct, with interlacing conducting paths to other neurons, and so far as we know, without actual anatomical union.

Many neurological phenomena are based upon the recognized trophic relationship of the cell to the axon and dendrites, for these processes degenerate if the cell become injured or diseased. If the process be severed the distal part degenerates. The cell atrophies to some extent if separated from its branches.

**Central and Peripheral Neurons.**—Cortical motor impulses, according to our conception, must pass from a motor cell in the cortex of the brain through its axons to, for example, a cell-body in the anterior horn of the spinal cord, the end brushes transferring impulses to the latter through the corresponding dendrites. The impulse here leaves the territory of the central or upper neuron for that of the peripheral or lower neuron, and is distributed by the latter in turn through its axon to the voluntary muscles, which it supplies.

In similar manner trophic, secretory and inhibitory impulses travel through their respective axons from their cell-bodies to the cells in the nuclei of the cranial nerves or the anterior horns of the cord. Functionally speaking, therefore, the cranial nerve nuclei are peripheral, although situated within the medulla.

The upper motor neurons exercise to some extent an inhibitory influence over the lower. The lower neuron is charged with the care of the muscle which it supplies, the nutrition, reflex irritability, and tone being dependent upon its integrity of function.

The path for the conduction of sensory impulses from the end organs in the skin or elsewhere lies through dendrites which bring these impulses to the cell, situated, for example, in one of the ganglia of the posterior roots of the cord. They are carried thence through the corresponding axon by way of the posterior root to the cord, to be in turn passed on through the upper neuron to the brain. It is thought that a third system of neurons conducts and distributes the impulses within the brain. Centripetal impulses are distributed, by a most complicated mechanism, to other neurons more or less related in function to the primary one, perhaps at a much higher level in the cord. Cells of neurons serving the same general function are grouped together forming nuclei which lie in the gray matter of the brain or cord, and their nerve fibers form definite tracts or bundles lying in the white substance or peripheral nerves. The intricate interrelation between sensation, special senses, secretion, motion, vasomotor control, and all the various functions demands a system of communication between different portions of the nervous system which constitutes the most complex, and, in its details, least comprehended field which we have to study.

**Segments of the Cord.**—The bundle made up of the anterior and posterior roots of one of the spinal nerves, with its mate upon the opposite side, and the portion of the cord to which they are attached, constitutes a segment of the cord. Immediately above and below lie the adjacent segments of the next superior and inferior spinal nerves. We have thus 31 segments, each being a unit possessing certain sensory, motor, vasomotor, trophic, and reflex functions, which it carries on by means of the anterior root fibers or motor fibers, and the sensory fibers of the posterior root upon each side. In the cervical region there are eight pairs of nerves and eight segments. Then come the twelve thoracic, five lumbar, five sacral, and one coccygeal pairs of nerves and segments. The segment, however, corresponds to the spinal nerve connected with it and not to the vertebra opposite which it is placed. The cord does not keep pace downward with the spinal column, so that the roots of the nerves travel downward to some extent before emerging.

In the cervical region we may thus locate the segment desired

by subtracting, according to Ziehen's rule, one from the number of the nerves, the remainder indicating the spinous process of the corresponding vertebra. In the region from the first to the fifth thoracic we subtract two, and in the remaining thoracic, three. Peripheral nerves generally receive axis cylinder processes from more than one segment of the cord, motion and sensation being both represented. Thus each segment is the seat of control of some definite aggregation of impulses originating, perhaps, a certain movement or movements, and localization of spinal disease depends largely upon the recognition of faulty action in the groups of muscles controlled by particular segments. For illustrative tables the reader is referred to the special works.

The cutaneous distribution of the peripheral nerves is well known and the segments of the cord corresponding to the distribution of the dorsal roots are fairly recognizable.

The following table modified from Butler represents the motor functions of the cranial nerves:

MUSCLES	
III. CRANIAL (MOTOR OCULI) .....	{ Sphincter iridis. Ciliary muscles. Levator palpebræ superioris. Rectus internus. Rectus superior. Rectus inferior. Obliquus inferior
IV. CRANIAL (PATHETICUS) .....	Obliquus superior.
VI. CRANIAL (ABDUCENS) .....	Rectus internus.
V. CRANIAL (TRIGEMINUS) .....	{ Associated movements of levator palpebræ. Muscles of the lower jaw.
VII. CRANIAL (FACIAL) .....	Facial muscles.
XII. CRANIAL (HYPOGLOSSAL) .....	Muscles of tongue.
IX. CRANIAL (GLOSSOPHARYNGEAL) )	{ Muscles of pharynx. Muscles of esophagus. Muscles of larynx.
X. CRANIAL (PNEUMOGASTRIC) ... )	
XI. CRANIAL (SPINAL ACCESSORY) .. )	

Hutchison and Rainy give the following table from which one may determine which muscles of the trunk and limbs are innervated by any given nerve:

UPPER LIMB		TRUNK AND LOWER LIMB	
POST-THORACIC....	Serratus magnus.	INTERCOSTALS..	Intercostals.
SUPRASCAPULAR....	Supraspinatus.		Rectus abdominis
	Infraspinatus.		External oblique.
EX. ANT. THORACIC	Pectoralis major (upp. part, low. part).	BRANCHES OF LUMBAR NERVES.....	Erector spinæ.
INT. ANT. THORACIC			Quadratus lumbo- rum.
MUSCULOCUTANE- OUS .....	Pectoralis minor.	GENITO-CRURAL.	Cremaster.
	Coracobrachialis.	ANTERIOR CRURAL .....	Sartorius.
	Biceps.		Pectineus.
SUBSCAPULAR .....	Brachialis anticus.		Rectus femoris.
	Subscapularis.	OBTURATOR....	Vastus externus.
	Teres major.		Vastus internus.
CIRCUMFLEX .....	Latiss. dorsi.		Crureus.
	Deltoid.	SMALL SCIATIC.	Gracilis.
	Teres minor.		Adductor longus.
MUSCULOSPIRAL....	Triceps.	SUP. GLUTEAL..	Adductor brevis.
	Ext. carp. rad. long.		Adductor magnus (with sciatic).
	Supinator long.	GREAT SCIATIC..	Gluteus maximus.
POST-INTEROSSEUS..	Supinator brevis.		Gluteus medius.
	Ext. carp. rad. brev.		Tens. vag. femoris.
	Ext. carp. uln.	INT. POPLITEAL.	Biceps femoris.
	Ext. comm. digit.		Semitendinosus.
	Ext. ossis metac. poll.		Semimembranosus.
	Ext. primi. intern. poll.	PLANTARS .....	Adductor magnus. (with obturator).
	Ext. secund. in- tern. poll.		Gastrocnemius.
	Ext. indicis.		Soleus.
	Ext. minimi digiti.		Tibialis posticus.
	Pronator radii teres.		Flex. comm. digit.
	Palmaris longus.		Flex. long. hallucis.
MEDIAN .....	Opponens pollicis.		Flex. brev. hallucis.
	Abductor pollicis.		Flex. brev. digit.
			Abductor hallucis.
			Adductor hallucis.
			Ext. brevis. digit.
			Interossei.

UPPER LIMB		TRUNK AND LOWER LIMB	
MEDIAN AND ULNAR (jointly)	{	Flexor longus pollicis.	{
		Flexor carpi radi-Ext. POPLITEAL	
		alis.	
		Flexor sublim. digit.	
		Flexor brevis pol- licis.	
ULNAR .....	{	Flexor carpi ul- naris.	{
		Adductor pollicis.	
		Muscles of little finger.	
		Interossei.	
			Tibialis anticus. Ext. prop. hallucis. Ext. digit. longus. Peroneus longus. Peroneus brevis.

### EXAMINATION OF THE PATIENT

Certain features of this examination have been considered in a previous section. In no department of medicine is an accurate history more essential, and in no department is the family history and a study of the idiosyncrasies, mental, and other peculiarities of the patient so essential. Many nervous diseases are familial, and many depend upon the inheritance of some special disease or tendency. Syphilis and alcoholism are of special import in this department of medical study.

In obscure cases our anamnesis must include the family history, a study of the patient's birth—possibility of injury from instruments, hemorrhage, etc.; his infancy—convulsions, night terrors; his school days—shyness, brightness, dulness, etc.; changes at puberty; occupation; sexual peculiarities and diseases; prolonged depressing diseases or experiences; use of alcohol and tobacco; and in general all departures from a normal life.

### MOTOR SYMPTOMS

After a history of the present illness is obtained we may start upon a systematic examination of the patient as regards motor symptoms. Paralysis of muscles indicates an affection of some part of the motor pathway, though not necessarily involving gross

structural damage. The patient is unable to move certain muscles or groups of muscles if the paralysis be complete; if partial, as is often the case, the movements are impaired. Movement may be absent or impaired on account of the patient's mental dulness in certain diseases without true paralysis, and on the other hand, movements may be defective on account of incoördination, as in locomotor ataxia, but not weak as in paralysis or paresis.

We test the power of the muscles by asking the patient to move the mouth, close or open the eyes, move the tongue, arms, legs, body, etc. The strength of the patient's hand is tested by comparing the grasp with that of the opposite hand or more exactly by the use of the dynamometer. The strength of the legs may be quite well determined by asking the patient to make certain movements which are resisted by the examiner.

Paralysis may be recognized in many instances by noting an abnormal position of the eye, drawing of the face to one side, dangling of the arm, wrist-drop, foot-drop, etc. The drooping of the toes and forepart of the foot signifies a peroneal paralysis; wrist-drop, paralysis of the musculo-spiral nerve; clawlike position of the fingers, ulnar paralysis; abduction and extension of the thumb or ape-hand, paralysis of the median nerve.

We should note especially as the patient moves whether the paralysis is flaccid or spastic. Since the trophic control of the muscles resides in the lower motor neurons in the anterior horns of the cord, an injury or disease affecting them brings about a flaccid paralysis of the muscles supplied, as in peripheral neuritis and anterior poliomyelitis. A destruction of the central neuron, as from hemorrhage, or injury to the brain, leads to a spastic condition of the muscles of the paralyzed part, as we see daily after cerebral hemorrhage. This is later often followed by a permanent contracture.

If the lateral tracts of the cord are involved we have a spastic condition of the muscles, as in primary lateral sclerosis, for the superior motor neurons make up the pyramidal columns. If the cord be entirely destroyed higher up, a flaccid paralysis results. The exact reason for this is not known.

Special names are used to designate the different localizations of paralysis. Hemiplegia signifies paralysis of one side of the body, commonly due to a lesion of the opposite side of the brain. The face escapes or is less affected than the limbs in most cases or, if affected, shows less permanent paralysis. A crossed hemiplegia designates a paralysis involving the face, ocular muscles, etc., on one side, and the rest of the body and limbs on the other.

Monoplegia is a paralysis affecting one member, even though not complete. Facial monoplegia is occasionally used to designate paralysis of one side of the face.

Diplegia is a double hemiplegia, rare in adults, but not especially uncommon in infants.

Paraplegia signifies a paralysis symmetrical in distribution and affecting the upper and lower limbs, although the common usage applies the term to the latter, unless the paralysis is designated as a brachial paraplegia when the legs are not involved.

The upper neuron type of paralysis may be due to damage either to the motor cells in the cortex, or the axons in any part of their course, including the terminal arborizations. Associated with the paralysis we have spasticity of the muscles, leading to contractures, exaggeration of the reflexes, normal reaction of the muscles to electrical stimulation, and no atrophy beyond that due to disuse. The muscles remain in good condition because they are still under the trophic control of their intact lower neurons, and, not degenerating, they react normally. The contractures are due to the influence of the stronger muscles in overcoming the weaker, though both are spastic. Thus we have the characteristic hemiplegic flexion of the toes, etc.

Lower neuron paralysis may be due to damage of any part of the lower neurons, cell-bodies, axons, or end brushes. The motor nuclei of the cranial nerves in the pons and medulla may be affected as well as the motor cells of the cord. The muscles involved are flaccid, become atrophied, lose their reflexes, and the reaction of degeneration may be found, for the trophic control of the lower neuron is destroyed.

Involvement of the motor cells in the cord gives the typical

paralysis of anterior poliomyelitis. Involvement of the axons in the peripheral nerves gives a paralysis of the type seen in toxic peripheral neuritis. In this type of paralysis, sensory disturbances are marked in contradistinction to that of anterior poliomyelitis. Reaction of degeneration is absent in purely muscular diseases.

The functional paralysis seen in hysteria may simulate almost any variety of organic disease. The absence of atrophy and of the reaction of degeneration and the preservation or exaggeration of the reflexes should put us on guard, although the contractures be present. Hemi-anesthesia, blunting of corneal and pharyngeal reflexes, other stigmata of hysteria, and the general circumstances of the illness should prevent error.

**Diagnostic Significance in Paralysis.**—Paralysis of cerebral origin is unilateral in most cases, excepting in case of the diplegias of childhood. No diagnosis of paralysis rests upon a good foundation until hysteria has been considered and excluded. There is less danger of inability to exclude it than of failure to consider it.

**FACIAL MONOPLÉGIA.**—This is the usual type of facial paralysis, the double form being rare. If of central origin it may arise from a lesion of the cortical center in the lower part of the anterior central convolution or in the nucleus of the facial nerve. Peripheral paralysis may originate in the nerve itself after it leaves the nucleus, in its course outward through the pons or within or outside of the bony canal.

**FACIAL DIPLEGIA.**—This is rare, since bilateral and symmetrical cerebral lesions must be present, excepting for the possibility of trauma of the two facial nerves at the same time. A tumor of the pons might press upon both nerves, or a lesion,—tumor, abscess, gummatous degeneration, etc., at the base of the brain might involve both nuclei or both nerves. Bulbar paralysis and diphtheritic neuritis must be considered.

**BRACHIAL MONOPLÉGIA.**—This is far more likely to be due to lesions of the brachial plexus or nerves of the arm, neuritis, than to be of central or spinal origin. If due to cerebral disease the focus must be a small and sharply defined one, hence hemorrhage and embolism are more likely to be the cause than thrombosis or injury.



A small tumor in the cortex or just beneath might be the cause. Lesions in the internal capsule are rarely circumscribed enough to give such a paralysis, since some fibers of the face or leg supply would almost certainly be compromised. A lesion in the cord would

require such an extraordinary distribution to paralyze one arm without other parts that it could rarely occur.

Gunshot and stab wounds involving the brachial plexus, or pressure from an aneurism or tumor may be the cause. Even then the paralysis is likely to be irregularly distributed, since some of the components of the plexus may escape. Neuritis is likely to involve only certain nerves, giving a partial brachial monoplegia, as the neuritis arising from pressure, crutch paralysis, or from lead, arsenical, or alcoholic intoxication. It is commonly bilateral, —bilateral brachial monoplegia. The pressure from crutches is more likely to give an even distribution of the paralysis than the toxic causes.

**CRURAL MONOPLÉGIA.**—It is possible to have this paralysis as the result of a limited cerebral lesion, comparable to that for the arm center described above. Such



FIG. 13.—ATROPHY OF ARM, FOLLOWING STAB WOUND, SEVERING UPPER TRUNK OF THE BRACHIAL PLEXUS. (Case of Dr. H. R. McGraw.)

origin is rare. A unilateral injury to the cord or a myelitis of similar distribution may cause a complete crural monoplegia. Anterior poliomyelitis is often the cause of the partial form, certain groups of muscles only being involved. A unilateral neu-

ritis from injury, pressure of tumor, bony disease, etc., should be considered.

**PARAPLEGIA** (crural).—This must be due to lesions of the cord or disease of the peripheral nerves. Perhaps the most striking form of spinal origin is that due to fracture of the spine, with destruction of the cord from pressure. Myelitis arising from Pott's disease, or otherwise, is a common cause. Hemorrhage into the cord, tumor of the cord, thrombosis, or embolism of the arteries, spinal meningitis, and the whole series of diseases involving the different columns of the cord may cause paraplegia. If the paralysis is spastic we expect a definite cord lesion. The flaccid type may be due to such a lesion, or some type of neuritis. Involvement of the bladder and rectal functions establishes the diagnosis of a cord lesion. The more exact diagnosis will be taken up in the consideration of the different diseases.

**HEMIPLEGIA**.—If organic in nature, hemiplegia is always due to a lesion in the brain. Complete hemiplegia,—arm, face, and leg of one side, is due to a lesion of the knee and anterior two-thirds of the posterior limb of the internal capsule upon the opposite side of the brain. The lesion may be small, for the motor fibers are closely packed together at this point. If hemianesthesia be present the lesion has encroached upon the posterior one-third of the posterior limb of the internal capsule.

In right hemiplegia, in the right-handed, we often have aphasia, indicating that the third left frontal convolution is thrown out of function by the lesion. If with hemiplegia we have oculomotor paralysis of the opposite side, the third nerve has been involved where it passes through the crus by a lesion affecting this area. Crossed paralysis, or hemiplegia with paralysis of the face upon the opposite side, signifies a lesion in the pons damaging the nucleus of the seventh nerve. Both nuclei may be involved, giving a double facial paralysis. Although the paralysis of the limbs in such a hemiplegia is of the upper neuron type, that of the face is of the peripheral neuron variety.

If with the hemiplegia we find anarthria and dysphagia we may expect to find the lesion in the medulla, involving the nuclei of some

of the last four cerebral nerves. The double hemiplegia noted as occurring in children is due to disease occurring upon both sides of the brain.

The most acute hemiplegias are due to injury, hemorrhage, and embolism. That from cerebral thrombosis may be rapid in development, but not commonly sudden. The slowly developed forms are generally due to the gradually increased pressure of a meningeal hemorrhage, tumor, gumma, gradual blocking of the vessels in syphilis, formation of an abscess, etc. In paresis, multiple sclerosis, and other degenerative types of brain disease hemiplegia may occur. It may be toxic or due to temporary circulatory changes in uremia.

### SENSORY SYMPTOMS

Dana classifies the sensory phenomena to be investigated, aside from the special senses, as follows:

TACTILE SENSE .....	{ Pressure Contact }	(Special Sense)	} Cutaneous sensations.
TEMPERATURE SENSE..	{ Heat Cold }	(Special Sense)	
PAIN SENSE .....	(General Sense)		Largely cutaneous, also muscular.
MUSCULAR SENSE...	{ Chiefly of weight }	(Special Sense)	} The power of coördinating muscular movements depends mainly upon these three special senses. <sup>1</sup>
ARTICULAR SENSE....	Of posture.. (Special Sense)		
TENDINOUS SENSE...	Of posture.. (Special Sense)		

The study of sensory disturbances is more difficult than that of the motor variety, because one is to some extent dependent upon the patient's statements and in the very cases needing investigation, these may be unreliable, and even wilful deception may need to be guarded against. The objective evidence obtained should be regarded rather than the subjective in cases of doubt.

**Paresthesia.**—As to the presence of numbness and tingling sensations, formication, burning sensations, and the long list of abnormal

<sup>1</sup> Quoted from Butler.

sensory phenomena pertaining to the skin and seen especially in those diseases implicating the posterior nerve roots and the peripheral nerves, we are almost at the mercy of the patient, since they cannot well be tested for objectively.

*Tactile sense*, either as to contact or pressure, is commonly tested by the use of the esthesiometer, the two points being applied to the skin to be examined, while the patient is blindfolded. If recognized as two separate contacts they are gradually brought nearer together until but one touch can be distinguished. Without the honest coöperation of the patient we can get no reliable results. One point may be felt as two, polyesthesia, or a touch upon one limb may be felt upon the other, allocheiria. The sensibility of the skin varies so widely in different individuals that we cannot be certain that our examination points to definite disease unless the variation from the table below is marked:<sup>1</sup>

Tip of tongue.....	1 mm. = $\frac{1}{16}$ in.	Back of hands.	30 mm. = $1\frac{1}{2}$ in.
Tip of fingers.....	2 " = $\frac{1}{8}$ "	Neck .....	35 " = $1\frac{3}{4}$ "
Lips .....	3 " = $\frac{3}{8}$ "	Forearm, leg,	
Dorsal surface of		dorsum of	
fingers .....	6 " = $\frac{1}{4}$ "	foot .....	40 " = $1\frac{3}{4}$ "
Tip of nose and fore-		Back .....	60-80 " = $2\frac{3}{4}$ - $3\frac{1}{4}$ "
arm .....	8 " = $\frac{1}{2}$ "	Arm and thigh	80 " = $3\frac{1}{4}$ "
Tip of toes, cheeks,			
eyelids, temple....	12 " = $\frac{1}{2}$ "		

Touch as distinguished from pressure may be tested for with a wisp of cotton in symmetrical locations in the usual way. The pressure sense may be tested by applying different weights, preferably of nearly the same size, but not of the same weight, to different parts, the limb or other parts tested being meanwhile supported to avoid confusion caused by muscular action. The pressure sense is more acute on the upper face, abdomen, and back of the hand than elsewhere. In testing patients with anesthesia we may ask them while blindfolded to touch a point which we have indicated by a touch just before. Normally the error should not be over two or three fingers' breadth.

<sup>1</sup> Butler: "Diagnostics of Internal Medicine."

The *temperature sense* is best tested by means of two test tubes filled with water, the one at a temperature of 60° F., the other at 100° F. The tubes are applied alternately or irregularly, and held in contact with the skin long enough to be sure that recognition is possible. The touch, heat, and pain sensations are commonly abolished together in those cases where we find tactile anesthesia. It is in syringomyelia, especially, that we note a disassociated sensory loss, pain and heat sensations being abolished while the tactile sense remains or is but little impaired.

*Pain* is tested for by means of a sharp point, as of a needle or pin, or roughly by pinching a fold of the skin. Especially with hysterical patients we should ask for a sharp distinction between touch and actual pain. Delay in feeling pain is common in tabes and in peripheral neuritis. Irregularly distributed anesthesia, or hyperesthesia, is often hysterical, though the hemi-anesthesia is more typical. Destructive organic disease involving a spinal segment or a nerve trunk may show an absolute loss of pain sense in certain areas. Hemiplegics may show anesthesia upon the affected side, due to the extension of the lesion in the internal capsule to the posterior third of the posterior limb.

The *muscular sense* is tested by giving the patient different weights to hold unsupported in the hand, or on the back of the foot. Normally any material difference in weight is easily detected.

The *articular and tendinous senses* are tested by putting one limb in a certain position and observing whether the patient can do the same with the opposite one.

**Ataxia.**—By this we mean a lack of ability to coördinate muscular movements. This coördination depends upon the integrity of the muscular, articular, and tendinous senses, for without their guidance the various muscle groups do not act in harmony and incoördinate movements result.

Ataxia of the upper extremities is tested by having the patient touch, with the eyes closed, the tip of the nose or other point, or more thoroughly by directing him to swing the two hands inward after extending the arms, and touch the points of the forefingers. In such motions as those of buttoning his clothing or writing, ataxia

may be detected. In the legs, if the patient be abed, we may find ataxia by directing him to touch one knee with the opposite heel. The Romberg test is better, the patient being directed to stand with his base of support narrowed by bringing the insides of the feet into contact, and then stand steadily with the eyes closed. A swaying to and fro of more than one inch is abnormal. The patient, if tabetic, may fall if the examiner be not prepared to support him.

Cerebellar ataxia is due to disease chiefly of the middle lobe of this organ and is not present when the patient is recumbent. Upon attempting to stand or walk, however, the defect in balancing power is shown by the staggering, so-called "titubating" gait, which is almost pathognomonic. The frequent preservation of the knee-jerks is notable.

**Astereognosis.**—This term signifies a defect in that sense by which we recognize solid objects by contact. Its presence is shown by the inability of the blindfolded patient to recognize a ball, knife, or coin by touch. It signifies disease in the parietal lobe, especially the superior parietal lobe.

**Aphasia.**<sup>1</sup>—Aphasia is inability to receive or communicate ideas, and is caused by a lesion in the cerebral hemispheres. It is not merely inability to talk, as this may be brought about by various forms of paralysis. It always involves loss of command of words in some form, whether as uttered, heard, seen, or written, and this loss is felt in internal speech.

In order to understand the symptoms of aphasia and their diagnostic significance we must know as far as possible the anatomy and physiology of the different parts of the brain concerned, especially of the centers of special sense. It is important to realize that the cells of sensory areas not only receive impulses causing the recognition of sensations, but that in them are stored the memories of sensations. There are special sensory areas called kinesthetic centers in which are stored the memories of sensations accompanying motions or following motions. If the kinesthetic memories corresponding to any particular motion are entirely absent, as from destruction of the center, that motion can not voluntarily

<sup>1</sup> The section upon Aphasia was written by Dr. Cyrus L. Pershing.

be initiated. If the memories are partially destroyed, either the motion cannot be initiated or is imperfectly carried out.

The precentral or Rolandic region is the motor area. At its lower end are located the areas for the face and throat, and here are initiated and coördinated such motions as chewing, swallowing, and whistling, but not those of speech. Acting through the face and throat centers and controlling them as far as speech is concerned is the special center of speech known as Broca's center, located at the posterior part of the third frontal convolution of the left side in right-handed people. In Broca's area are stored the memories of the complicated motions required for utterance, and here also such motions are initiated. Another special motor center is the writing center located in the second frontal convolution on the left side in right-handed people. Here are stored the memories of the motions used in writing, and this center controls the arm and hand centers of the Rolandic region in writing, as Broca's center controls the centers of face and throat in talking.

A special sense area is that of hearing located in the first and second temporal convolutions of both sides. In this area on the left side is the auditory speech center where the memories of spoken words are stored. The special sense area for vision is located in the occipital lobes. Here all visual sensations are perceived and all visual memories are stored except those for words. The visual center for words, where memories of printed and written words are stored, is located in the angular gyrus of the left parietal lobe.

In order to talk we must have ideas or concepts. A word is a spoken, written or printed symbol of a concept. A concept is formed in the mind by the association of sensations and the memories of sensations stored in the corresponding centers, brought about by the nervous tracts connecting them. The formation of a concept depends, therefore, on the integrity of the centers and their connecting tracts. For this reason the sensory centers are called conceptual or ideational centers. When the tone of a bell is heard the auditory center stimulates the visual center and its memories through the corresponding association tract and we have a visual image of a bell giving about that tone. Then the touch centers

may be aroused and we conceive how such a bell would feel, as rough, smooth, hot, cold. In a similar way we recall the sound of the word "bell" and the appearance of the written or printed word, and finally the efforts or motions required to speak or write the word. In this way the concept "bell" is formed, which would not be possible if the centers or tracts concerned were damaged or not functioning properly. Starr<sup>1</sup> gives a diagram illustrating this process. (See Fig. 14.)

**APRAXIA.**—Any disease which damages these centers, the tracts leading to or from them, or their association tracts, may cause apraxia, which is an inability to form concepts or to recognize objects presented to consciousness by one or more of the senses. Apraxia includes aphasia. Aphasia is part of apraxia or a special form of apraxia. In order to discuss aphasia some further definitions are necessary.

**Mind-blindness or visual apraxia** is an inability to recognize things seen, and always includes words. The lesion that produces it is almost necessarily bilateral.

**Word-blindness** is an inability to recognize words seen. It is a part of mind-blindness, but may occur independently of mind-blindness. The printed or written words of the patient's own language mean no more to him than those of a language he never heard of. The lesion causing it is in or near the left angular gyrus.

<sup>1</sup> Starr, M. Allen: "Organic and Functional Nervous Diseases," 1909.

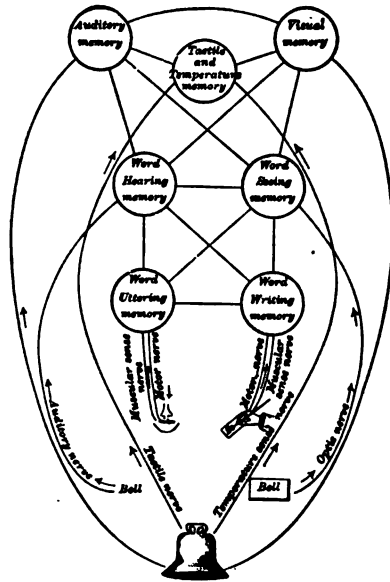


FIG 14.—DIAGRAM TO ILLUSTRATE THE CONCEPT "BELL" AND TO SHOW THE VARIETIES OF APRAXIA AND APHASIA. The memory pictures are relics of past perceptions received through different senses. Their association makes up the mental image "bell." The word image is made up of the memories of the sound and appearance of the word, and of the uttering and writing-effort memories; these are joined together. The mental image and the word image are also joined with one another, making up the concept "bell." (Charcot.)



Mind-deafness or auditory apraxia is inability to recognize things heard. It always includes words. The lesion is almost necessarily bilateral.

Word-deafness is an inability to recognize words heard. It is present in all cases of mind-deafness, but may occur independently of mind-deafness. The words of the patient's own language sound like foreign words to him. The lesion causing it is in or near the first and second convolutions of the left temporal lobe.

Alexia is inability to read, often secondary to word-blindness or word-deafness.

Agraphia is inability to write. It may be due to lesion in the writing center or be secondary to other lesions.

Paraphasia is inability to talk properly. Words are misplaced in the sentence and syllables are misplaced in words so that the talk becomes a mere jargon in some cases.

Paralexia is inability to read aloud, similar to paraphasia.

Paragraphia is inability to write because of words and syllables being misplaced.

Verbal amnesia is inability to recall a word when the idea or the rest of the concept is in the mind, the word being recognized as soon as heard.

**MOTOR APHASIA.**—Motor aphasia is caused by a lesion in Broca's center—cortical motor aphasia; in the white matter beneath it connecting it with the lower centers,—sub-cortical motor aphasia; or in the fibers connecting it with other centers,—transcortical motor aphasia or conduction aphasia. In complete destruction of the center the onset is generally sudden. At first not even gestures can be used, and no words, or only a very few can be used, these usually expletives or exclamations. The patient knows what he wants to say and recognizes his mistakes because the other concept centers and their connecting tracts are intact, but he is unable to remember and initiate the movements necessary for utterance. The patient understands what is said to him, but of course is unable to read aloud. He is also unable to read quietly to himself. Although he sees the word perfectly he is unable to associate with it the concept corresponding to it without the aid of the motor speech center.

Normally in reading the visual center stimulates the auditory center and we imagine the sound of the word, then both centers stimulate the motor speech center, and we imagine the movements necessary to speak the word. Not until then are the other idea centers aroused and the conception of the word formed. If an important link in the chain, like the motor speech center, is missing, the concept is not formed. For the same reason the patient is unable to write. Very rarely in a case of this kind the patient is able to read quietly, and this can only be explained on the hypothesis that owing to some personal peculiarity the visual center alone can arouse all the other centers concerned in the idea. The recovery is only partial and very slow, a matter of years, and is probably due to the education of the motor speech center on the right side. In cases of injury or partial destruction of the center, the symptoms are less extensive—more words are retained, and the recovery is more rapid. This condition is distinct from aphonia or anarthria, due to the functional or organic paralysis of the muscles of speech. In motor aphasia the ordinary motions of face, tongue, and lips are undisturbed.

The lesion of subcortical motor aphasia is usually in the internal capsule, and the symptoms are therefore accompanied by hemiplegia. Reading and writing are not interfered with for the reason that, the center being intact, the motor speech memories do their part in stimulating the other centers and thus forming the concept. Recovery is very rapid as compared with the cortical variety, probably because the motor speech center on the right side, which is connected through the fibers of the corpus callosum with the trained center on the left, is able to take up the work more quickly. There are as many kinds of transcortical motor aphasia as there are tracts from the other idea-centers to the motor speech center. The cases are rare. Wernicke's conduction aphasia is caused by interruption of the fibers connecting the motor speech center with the auditory center. Its symptoms are loss of imitative speech, paraphasia, agraphia, paralexia on reading aloud, and loss of comprehension of print. There is no mind-blindness nor word-blindness.

**AUDITORY APHASIA.**—Auditory aphasia is caused usually by a

lesion in the auditory cortex for words in the left temporal region. The symptoms vary somewhat according as the destruction is complete or partial. In complete destruction there is absolute word-deafness and auditory amnesia. There is loss of spontaneous speech or paraphasia, alexia, or paralexia, and agraphia or paragraphia. The auditory memories are the most important factor of the word concept. In learning to talk, read, and write, the development of the word-hearing center precedes that of all the others, and for proper functioning the others are dependent on it. The interference with speech is due to the fact that the motor speech center cannot initiate and coördinate the separate movements necessary to pronounce a word unless the mental image of the sound of the word is in the mind. The cause of the difficulty in writing is the same. Such a patient's talk is jargon, and he does not recognize his mistakes as does the motor aphasic, because he is word-deaf. The inability to read is due to the fact that without the auditory memory of the word the word-concept is imperfect.

Some patients who rely more on their visual memories of words in forming word-concepts have less disturbance of the above functions than the average person would have. This condition might account for the ability to read aloud without understanding what is read that some patients with auditory aphasia possess.

A lesion in the white matter just below the word-hearing center would produce word-deafness without verbal amnesia. Such a patient may be able to talk correctly, and there is no paragraphia, though writing from dictation is impossible on account of the word-deafness.

An interruption of the intercortical fibers between the word-hearing center and the visual center produces a form of aphasia in which, when an object is seen and its use recognized, its name can not be recalled, although the name may be recognized when heard. Also the name is recalled often when the object is handled, tasted, or smelled. Similarly if the name is heard alone the appearance of the object can not be recalled. This form of aphasia is sometimes a localizing symptom in cases of brain abscess in the temporal region following middle ear disease, or from other cause.

If one or more of the transcortical tracts connecting the word-hearing center with the other sensory centers is broken, we have varying degrees of auditory amnesia. After exhausting diseases, in migraine, sometimes after an epileptic convulsion, and even in conditions of normal weariness this is a fairly common temporary functional symptom, caused by the association tracts sharing in the general exhaustion or fatigue of the brain.

**VISUAL APHASIA.**—Visual aphasia caused by disease of the cortex of the left angular gyrus is characterized by word-blindness and alexia. A lesion of the left angular gyrus usually extends inward far enough to involve the fibers of the left optic radiation, and, therefore, accompanying it, there is defect in the right visual field if not hemianopsia. Spontaneous speech is interfered with, for although the number of words that can be correctly used is large the names of objects seen can not be recalled and the use of nouns is thus limited. If mind-blindness is not present, i.e., if the visual areas on both sides of the brain are not involved, an object that cannot be named may be fully recognized and its use described. Often the name can be recalled if the auditory center and motor speech center are aroused through another sensory path, as by touch, taste, or smell. Writing is impossible either spontaneously or from dictation or copy, as the memories of the appearance of words and letters are destroyed. In some cases figures are remembered better than words, and games like cards can be played.

In subcortical visual aphasia the incoming fibers are interrupted before they reach the cortex of the left angular gyrus, consequently, although words seen are not recognized, the visual word memories are not interfered with. Although print and writing cannot be read or copied, spontaneous writing and speech are not interfered with. Thus, in some cases the patient may write correctly and then not be able to read what he has written.

**GRAPHOMOTOR APHASIA.**—Graphomotor aphasia, the inability to write on account of lack of coördination of the necessary movements of the right hand and arm, may be caused by disease at the foot of the second frontal convolution. Agraphia, however, is usually a symptom accompanying some other form of aphasia.

The above is a mere outline of the main symptoms of aphasia. If the fundamental facts are understood and the symptoms correctly elicited, the diagnosis in any particular case can be reasoned out with a fair degree of accuracy. It is impossible to go into all the details here. With so many centers and their connecting tracts involved the combinations that can occur are of course numerous. Also the number and severity of symptoms vary with the extent and severity of the disease.

It is to be remembered that while the speech centers are in the left cerebral cortex in right-handed people they are in the right side in left-handed people. Consequently, in the left-handed aphasia goes with lesions of the right hemisphere.

A temporary functional aphasia is often found in such diseases as epilepsy, migraine, aural vertigo, and toxemias, such as anemia, alcoholism and uric acid diathesis, as well as after exhausting diseases.

In the examination of an aphasic patient, the following information should be elicited: If there is difficulty in speech, is it due to peripheral paralysis or to disorder in the higher centers or their association tracts? Is the number of words used very limited? Are words and sounds misplaced and wrongly used so that the talk is senseless? Does he understand what is said to him? Does he answer questions intelligently? Does he respond to unexpected requests without any gesture or sign to help him? Can he select familiar objects when named? Can he give the name of objects seen, heard, felt, tasted, or smelled? If there is difficulty in meeting the above tests, can it be due to deafness? Does he recognize and understand the nature of objects seen? Can he read and understand what he reads? Can he read aloud? Does he respond to written requests? If there is any difficulty is it due to loss of vision? Can he express himself in writing? Can he copy? Can he write from dictation? Can he write the names of objects seen, heard, felt, tasted, or smelled?

**SIGNIFICANCE OF SENSORY DISTURBANCE IN DIAGNOSIS**

**Hyperesthesia.**—A hypersensitiveness to touch and other impressions is common. If generally distributed and associated with other symptoms it suggests meningitis. Serious organic nervous lesions may be accompanied by hyperesthesia, as brain tumor, hemiplegia (on the paralyzed side), locomotor ataxia, etc. Above the level of the anesthesia, in unilateral lesions of the cord, on the side of the lesion, is a narrow band of hyperesthesia. In migraine, in many cases of neuritis, and in association with many forms of neuralgia, we may note increased sensitiveness of the area involved. The general or localized hyperesthesia noted in infectious fevers,—typhoid, influenza, and in conditions of disturbed nutrition,—anemia, alcoholism, etc., may perhaps more often than otherwise be attributed to an ill-defined peripheral neuritis.

Perhaps the most important type of hyperesthesia for the clinician is that associated with hysteria. Its varieties are so numerous that any form of this sensory disturbance not readily classified should be assumed to be hysterical until proved otherwise. The hyperesthetic areas over the lower abdomen in young women, hysterogenic zones, pressure upon which may originate or check hysterical convulsions, the oversensitiveness of the spine, of portions of the surface of the trunk, scalp, face, etc., should be noted. The hysterical breast, a painful and excessively tender mammary gland, commonly in neurotic young women, deserves especial mention. It is an open question whether the tenderness of Valleix's points in intercostal neuralgia may not be attributed to hysteria as often as neuritis.

**Referred Pains.**—In the study of visceral diseases we must note the occurrence of referred pains. Head's exhaustive researches make it plain that pain in a given organ is often accompanied by pain and tenderness in a certain distant area of the skin. It is probable that an organ possessing but little sensibility, by the sending of its sensory impulses to its spinal segments, and the transference of these impulses to a more sensitive area (skin) supplied from the same segment, may cause much more discomfort in the

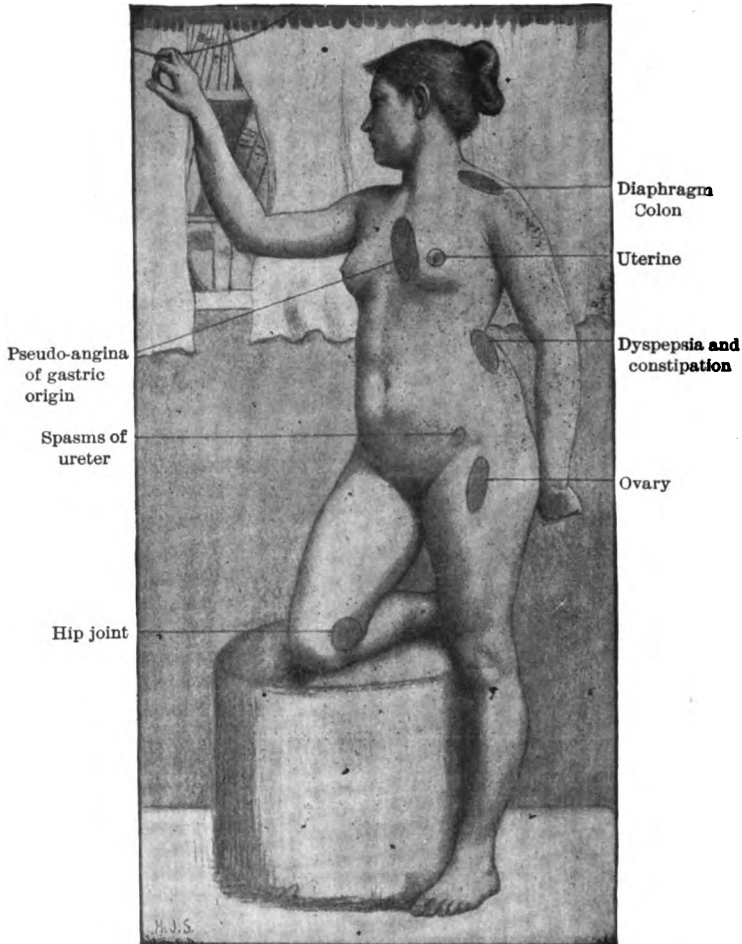


FIG. 15.—THE LOCATION OF TRANSFERRED PAINS (Dana). Figure redrawn in charcoal after an imported photograph. These pains, through the existence of more or less indirect or roundabout sensory nerve connections, are felt at points of varying remoteness from the source of the irritation which produces them. Thus, pain in the knee may be due to disease of the hip joint, the lesion affecting one of the terminations of the nerve much nearer to the origin of the latter than that in which the pain is felt; or the mammary gland may swell and become painful when a distant organ—the uterus—is the seat of a pain-producing process. (Butler's "Diagnostics of Internal Medicine.")

secondary area than the patient feels in the real seat of the disease. (See Fig. 15.)

In many diseases the pain is felt at a region distant from that of the greatest pathological change, through the transference of the

sensation peripherally in the nerve system involved, as in the knee pains of hip disease, or the radiating pains when one branch of the trifacial is involved. If the pain be intense it may overflow into neighboring channels, so to speak, as in the arm pains of angina pectoris.

In compression of the spinal cord there may be found areas devoid of tactile and pain sense, but which are yet acutely painful,—*anesthesia dolorosa*.

**Anesthesia.**—This phenomenon, like hyperesthesia, is so commonly hysterical in origin that it should suggest functional disease to the medical mind rather than organic, because of the predominance of functional disturbance of this nature. While literally denoting loss of tactile sensibility, it is commonly employed clinically to denote loss of sensation in general.

By hemianesthesia we mean loss of sensibility in one lateral half of the body, often accompanied by loss of special sensation upon that side. If the patient be a young female and the phenomenon be noted upon the left side, we may assume it to be a manifestation of hysteria unless signs of organic disease be found, for this is the most common cause of hemianesthesia, and is more frequent upon the left side. One must beware of a decision which excludes hysteria even when the right side is involved, and even in the male sex. Loss of corneal and pharyngeal reflexes should be looked for as confirmatory signs in the diagnosis.

Next in frequency is the generally incomplete hemianesthesia of organic hemiplegia, due to involvement of the posterior portion of the posterior limb of the internal capsule, as noted elsewhere, the sensory fibers being damaged by the hemorrhage, tumor, or other lesion causing the hemiplegia of the same side. If with hemiplegia and hemianesthesia there be oculomotor paralysis of the opposite side, the lesion is to be sought in the crus of that side. Crossed facial anesthesia is occasionally seen in lesions of the pons or medulla below the exit of the fifth nerve. In unilateral damage to the cord we may find incomplete anesthesia with analgesia upon the side opposite the lesion, with partial hemiplegia of the injured side. Various obscure lesions in the brain may be accompanied by hemi-



anesthesia without the exact topical diagnosis being readily possible. If the anesthesia be bilateral and affecting the lower limbs, or these in conjunction with the lower part of the trunk, we may assume a cord lesion of almost any origin,—fracture, compression from Pott's disease, pressure from tumor, hemorrhage, myelitis, etc., to be the cause if hysteria be excluded, since brain lesions are practically out of the question. The presence of sphincter disturbance and bed-sores will be confirmatory of organic lesion, while in hysteria the sensation often remains intact over the genital and lower sacral region.

Irregularity in distribution of anesthesia is commonly significant, according to the distribution, of either some form of neuritis or of hysteria. The characteristic feature of hysterical anesthesia lies in the utter disregard of the anatomy of the nerve supply of the affected region, and of the segmental representation. If by comparison of the mapped-out area with the figures we find a lack of such correspondence, we assume that the anesthesia is hysterical, bearing in mind, however, the possibility of trauma or disease of several different nerve trunks or branches, which might give an appearance, upon superficial examination, of lack of the correspondence noted. "Patchy" anesthesia is practically always neurotic in origin.

**Hyperalgesia.**—By this term we mean excessive sensitiveness to pain, analgesia signifying a loss of such sensibility. The former is sufficiently considered under hyperesthesia, of which it may be considered a part. Analgesia is especially significant in the diagnosis of syringomyelia, yet hysteria is so enormously more frequent as a cause that it must always be considered. The so-called Morvan's disease and syphilis must also be considered.

The study of the disturbance of the temperature sense is of especial use in the diagnosis of syringomyelia, in which there is often loss of sensation for heat or for cold, or reversal of these sensations. In tabes a less pronounced disturbance may be noted.

The involvement of the tendinous, muscular, and articular senses is the basis of ataxia excepting in the cerebellar form, where the lesion is central, while in the ordinary form it is due to interruption

of the passage of such sensations along the sensory conducting tracts. Locomotor ataxia is the disease in which it is seen to best advantage, although the tracts may be interrupted by lesions in the pons, crura, and regions above, as in hemiplegia. In multiple neuritis the damage is rather peripheral, while in injury or disease of the cord the interruption is in or near the cord. In various nervous diseases any one or more of these senses may be involved or lost.

### THE REFLEXES

Each segment of the spinal cord, as we have seen, is the center for the operation of certain groups of muscles. It is also the center for certain movements involving the action of these muscles, and spoken of as reflexes. A stimulus from a sensory nerve comes to a center in the cord, passes over to the corresponding motor center, and is reflected out upon the motor nerves to the muscles supplied, giving rise to a so-called reflex contraction. The whole path is called the reflex arc, and a break in either afferent, central, or efferent portion of the arc abolishes the reflex. As we have seen elsewhere, the brain sends fibers to each segment, having the function of inhibiting the reflex arising there. Irritation increases the inhibitory action of these fibers tending to decrease or abolish the reflex, while destruction of course withdraws their influence and the reflex becomes exaggerated. Several motor impulses at different levels may be excited by a single sensory impulse.

The reflexes are cutaneous—superficial, or tendinous—deep. The organic (excito-reflex) actions pertain to the functions of defecation, respiration, urination, etc., and have been considered elsewhere. Each has a special center in the gray matter of the cord and responds in its special manner to a certain stimulus.

#### LOCALIZATION OF REFLEX ACTS IN THE SPINAL CORD

REFLEX ACTS	LOCALIZATION IN SEGMENT
Pupillary reflex through the sympathetic: Dilation of the pupil produced by irritation of the neck.	Fourth cervical to first dorsal.

REFLEX ACTS	LOCALIZATION IN SEGMENT
Scapular reflex: Irritation of the skin over the scapula produces contraction of the scapular muscles.	Fifth cervical to first dorsal.
Biceps and supinator longus: Tapping their tendons produces flexion of the forearm.	Fifth and sixth cervical.
Triceps reflex: Tapping tendon produces extension of forearm.	Sixth cervical.
Scapulohumeral reflex: Tapping the inner lower edge of the scapula causes adduction of the arm.	Seventh cervical.
Tapping extensor tendons at the wrist causes extension of the hand.	Sixth to eighth cervical.
Tapping flexor tendons at the wrist causes flexion of the hand.	Seventh to eighth cervical.
Palmar reflex: Stroking palm causes closure of fingers; finger clonus.	Eighth cervical to first dorsal.
Abdominal reflex: Stroking side of abdomen causes retraction.	Ninth to twelfth dorsal.
Genital reflex: Squeezing the testicle causes contraction of the abdominal muscles.	First to third lumbar.
Patella tendon: Striking tendon at knee causes extension of the leg; "knee-jerk."	Second and third lumbar.
Achilles tendon reflex: Tapping the Achilles tendon causes flexion of ankle.	First to third sacral.
Foot clonus: Extension of Achilles tendon causes flexion of the ankle.	First to third sacral.
Plantar reflex: Tickling sole of foot causes flexion of the toes.	First to third sacral.

REFLEX ACTS	LOCALIZATION IN SEGMENT
Babinski's reflex: Scratching sole of foot causes extension of great toe and flexion of the others.	
Mendel's reflex: Tapping the tendons of the toes causes flexion or extension of the toes. <sup>1</sup>	
Epigastric reflex: Stroking breast causes dimpling of the epigastrium.	Seventh to ninth dorsal.
Cremasteric reflex: Stroking inner side of thigh causes retraction of scrotum.	First and second lumbar.
Gluteal reflex: Stroking buttock causes dimpling in the fold.	Fourth to fifth lumbar.

**Superficial Reflexes.**—These are much inferior in importance to the deep reflexes. Depending upon the medulla, we have: (1) the conjunctival reflex—the eyelids close suddenly through the action of the facial nerve upon the orbicularis palpebrarum, upon irritation of the conjunctiva or the stimulation of a bright light; (2) the pupillary skin reflex—stroking or pinching the skin of the side of the neck causes the pupil to dilate; (3) palatal—irritation of the palate causes it to retract (often lost in hysteria and bulbar paralysis).

From the cord we have: (1) epigastric reflex—the epigastrium retracts upon the same side if the chest be stroked downward from the nipple; (2) abdominal—the abdominal muscles contract on the same side upon stroking downward from the rib margin in or near the line of the nipple and reflexes may be observed at several different levels; (3) cremasteric—the testicle retracts from pinching or stroking the skin of the upper and inner aspect of the thigh of the same side. In the female the response may constitute the inguinal reflex, due to contractions of the fibers of the internal oblique above Poupart's ligament; (4) plantar—on tickling the sole of the foot all the toes are flexed; (5) anal—the sphincter contracts

<sup>1</sup> Quoted from Starr.

if the skin of the anus be irritated. The gluteal and scapular reflexes have but little significance and are frequently missing.

The skin reflexes in general are so often absent and vary so much if present that little excepting the functional integrity of the reflex arc can be deduced from an examination of any of them, unless the response is decidedly increased.

**Tendon, Joint, and Periosteal Reflexes.**—These are of much greater significance than the foregoing. Roughly, in order of importance, clinically, they are as follows:

**KNEE-JERK** (patellar tendon reflex).—The knee is supported across the other knee or on the arm of the examiner, so that the muscles may entirely relax. The tendon is struck with the edge of the hand, or the percussion hammer. The movement of the foot, due to extension of the leg, is the response commonly looked for. The contraction of the extensors of the thigh, as noted by sight or feeling, should be the criterion; for in conditions of weakness no perceptible movement of the foot may occur, notwithstanding the muscles have responded by contraction. If not found the “reinforcement” procedure should be tried—the test being made while the patient firmly grasps the arms of the chair with the hands, or, hooking the hands together, makes an effort to pull them apart. The same phenomenon may be induced by pulling the patella downward and then tapping the quadriceps extensor directly. If the knee-jerk be greatly exaggerated we may obtain a knee clonus by pushing the patella downward suddenly and holding it down while the leg is extended.

**ANKLE-JERK** (Achilles tendon reflex).—This is excited by rendering the calf muscle tense by dorsally flexing the foot and then tapping upon the tendon, and may best be performed while the patient kneels in a chair.

*Ankle Clonus.*—In association with increased knee-jerk we often find this phenomenon. With the leg relaxed the examiner pushes the toe of the foot upward while supporting the calf of the leg. The foot, owing to the contraction of the calf muscles, pushes against the hand in a series of jerks, if the clonus be present—Clonic spasm of the Soleus. A similar but less characteristic phenomenon may

be induced in hysterical patients, but is distinguished by the presence of other stigmata of hysteria, and by the absence of organic disease.

**BABINSKI REFLEX.**—With the foot supported at rest in such a way that the sole is exposed, the skin is irritated by stroking with a match, blunt pin, etc., from the heel forward to the ball of the great toe, unless it is excited with a lesser irritation, the response, if positive, being noted in a dorsal extension of the great toe, and often of some of the others. In the normal patient beyond the age of infancy plantar flexion is the rule. The presence of the Babinski reflex is looked upon as almost positive evidence of a lesion in the pyramidal tracts. Its absence in hysteria is frequently of much aid in diagnosis. Absence of plantar reflex with exaggerated knee-jerk is characteristic of hysteria.

**OPPENHEIM'S REFLEX.**—This consists in a retraction of the great toe and end of the foot due to contraction of the tibialis anticus, induced by stroking the inner surface of the leg near the tibia, or by "straddling" the tibia with the thumb and finger, and stroking downward. It is confirmatory when the tendon reflexes are exaggerated. Gordon's reflex has a similar meaning—extension of the toes by deep pressure upon the calf muscles. It is often positive when the tendon reflexes are exaggerated.

**JAW-JERK.**—The finger placed upon the chin is strongly percussed while the mouth is held half open. If present the jaw-closing muscles will contract.

**ELBOW-JERK.**—With the arm hanging downward from the supported and flexed elbow the triceps tendon is strongly tapped. The forearm is extended by the contraction of the triceps if the reflex be present.

**WRIST-JERK.**—The hand hanging downward from the supported wrist extends suddenly if the extensor tendons be tapped above the wrist joint.

The radial (periosteal) reflex is elicited by tapping the styloid process of the radius. Flexion and pronation of the hand and slight flexion of the elbow may be noted.

**BICEPS REFLEX.**—The biceps reflex consists in a flexion of the

forearm upon striking the biceps tendon. Practically all accessible muscles will yield reflexes under some conditions.

**Significance of the Deep Reflexes in Diagnosis.**—Any of them may be absent in health, the most important, the knee-jerk, being more constantly present. In adults in whom this reflex is normally absent, it is probable that some forgotten nervous disease of infancy or childhood may have led to its abolition.

A lesion of any part of the reflex arc may lead to the disappearance of the reflex and therefore the presence of a normal reaction signifies a normal condition of the afferent and efferent nerves involved, of the posterior roots and anterior horns of the cord, and of the muscles affected. Absence of reflex signifies either (1) disease of the sensory nerve, as in neuritis; (2) of the posterior roots and columns, as in tabes; (3) of the anterior horns, as in anterior poliomyelitis; (4) or of the motor nerve or the muscle. The common absence of the tendon reflexes in diabetes, and after severe diphtheria, in spinal meningitis, and after epileptic convulsions, or apoplectic stroke, should be noted. Absence of ankle-jerk is sometimes found before loss of knee-jerk in tabes, and has the same significance, though of less importance.

**Exaggeration of Tendon Reflexes.**—This signifies that the inhibitory impulses coming from the brain in health through the lateral columns are blocked, and that therefore the sensory impulses coming to the cord from without bring about a sharper reflex action than normal. The presence of the Babinski reflex, common under these conditions, is to be regarded as pathognomonic of disease of the lateral columns of the cord or motor tract. The reflex centers in the cord may be abnormally irritable under some circumstances, apart from the abolition of the inhibitory impulses mentioned. The knee-jerk may be exaggerated shortly, not immediately, after the occurrence of cerebral hemorrhage, on the affected side, and in hereditary cerebellar ataxia, general paresis, and cerebellar paralysis of children.

Spinal causes of exaggerated knee-jerks are notably lateral sclerosis and *amyotrophic* lateral sclerosis, often preceded in the latter disease by exaggerated arm reflexes which disappear as the disease progresses. Any process in the cord interfering with the transmis-

sion of motor impulses from the brain may cause exaggerated reflex action as in syphilitic paraplegia, injuries, myelitis, multiple sclerosis, syringomyelia, etc. If the lesion be unilateral, the increased reflex is on the affected or paralysed side. Increased knee-jerk is extremely common as a functional affection in hysterical and neurotic individuals, in tetanus, and after the free administration of strychnin.

### ELECTRODIAGNOSIS

For neurological diagnosis a galvanic current apparatus is more useful than the faradic, but both are essential for thorough work. In cities the apparatus is commonly operated by the use of the light or power current supplied, with suitable reducing, measuring, and safety devices. Paralyzed muscles are first tested with the Faradic current. The large or indifferent electrode is placed upon the sternum or abdomen and the small one upon the motor point of the muscle to be tested, gradually increasing the current until at the opening of the primary circuit the muscle contracts, or the application becomes painful. A normal muscle should respond with a quick contraction, the contraction continuing during the passage of the current and being within certain limits, in proportion to the strength of the current. The contraction becomes tetanic if the interruptions are sufficiently frequent.

The response of the muscle is diminished more or less according to the degree and age of the pathological process in inferior neuron motor paralysis (neuritis, poliomyelitis, etc.), the response disappearing at the end of a fortnight in the average case. The probable extent of the paralysis in these acute processes may be judged to some degree by the diminution in the faradic contractility. The faradic current is not strictly a current, and it matters not, as far as polarity is concerned, which electrode is applied to the nerve.

The galvanic current upon the other hand depends for its action upon which pole is applied to the nerve, and whether the circuit is opened or closed. With a gradually increased current we have, with the cathode applied over the nerve, at first a contraction only upon closing the circuit (CCC). As the strength increases we next have



a contraction upon closing the circuit with the positive pole on the nerve (ACC). Next comes the contraction on the positive opening (AOC), and lastly, often not until the current is painful, the contraction upon negative opening (COC). With fairly strong current the two intermediate reactions may be reversed. We may express the order of the above reactions of muscles to galvanic current as follows:  $CCC > ACC > AOC > COC$ .

**Reaction of Degeneration.**—When degeneration is taking place in a muscle from trauma or disease affecting its nerve supply (neuritis, poliomyelitis) the faradic reaction becomes sluggish or disappears. At the same time the galvanic reactions change so that the response of the muscle is slow and sluggish, and the anodal closing contraction (ACC) becomes greater than the cathode closing contraction (CCC), although both are less vigorous than the reactions of a normal muscle. The anode opening contraction (AOC) disappears and the cathode opening contraction (COC) is present in but few cases.

If the paralysis is temporary and recovery sets in, these reactions gradually return to normal. If it be permanent the galvanic reactions eventually disappear (1 to 2 years).

The information we obtain from electrodiagnosis may be epitomized as follows: If muscles respond properly to faradic and galvanic currents, neither they nor their lower (motor) neurons are diseased, but the central nervous system may be. If the reaction of degeneration is present, disease of some part of the lower neuron is positively indicated.

### TROPHIC AND VASOMOTOR DISTURBANCES

In certain conditions the chief manifestation lies in a disturbance of the nutrition of the parts involved because of the lesion of the nervous structures which preside over nutrition. We speak of these as trophic diseases. The trophic influences travel chiefly in the motor nerve fibers to the muscles, but there may be separate fibers yet unknown elsewhere. The rapid wasting in poliomyelitis is known to be due to the loss of the trophic influences of the lower neurons. Cortical trophic centers are believed to be at fault as the explanation of the slow atrophy in the case of old central paralysis. The atrophy

which accompanies amyotrophic lateral sclerosis and bulbar palsy is similar to that of chronic poliomyelitis; but the gray matter of the cord is found to be involved, the upper and lower neurons being both implicated, the latter being responsible for the atrophy.

The unused muscles in joint disease, etc., shrink uniformly, but do not degenerate, and retain their electrical reactions. In so-called "arthritic atrophy" this process is thought to be reinforced by reflex irritation proceeding from sensory nerves about the joint.

In the differential diagnosis of the muscular atrophies, the presence of the reaction of degeneration and of fibrillary twitching is characteristic of the spinal and neuritic types, the absence of both, of the idiopathic muscular atrophic type (muscular dystrophies).

Muscular hypertrophy occurs as a trophic process in Thomsen's disease. In pseudohypertrophy the increase is due to proliferation of interstitial tissue and fat, and not to increase in the muscle fibers.

Certain internal secretions have a trophic influence, and the withdrawal or derangement of the secretion, due to disease of the glands (thyroid, pituitary) may cause the changes in the skin and other tissues noted in such diseases as myxedema, acromegaly, and adiposis dolorosa.

An increase or decrease of the blood supply beyond the normal limits, due to disease of the vasomotor mechanism, is the essential cause of such conditions as Raynaud's disease, erythromelalgia and angioneurotic edema. Angioparalysis is the active agent in some of these troubles, and angiospasm in others. In women at the menopause, in exophthalmic goiter, and in neurasthenia, an ataxia of the vasomotor apparatus (angioataxia) is often seen, with marked alternations between spasm and relaxation of the vessels.

Trophic disturbances are notoriously frequent and troublesome in many serious nervous diseases. As examples, we may mention the bed sores of paraplegia, the Charcot joints in tabes, the arthropathies of myelitis, and the osteoarthritis of various chronic cord diseases. The painless whitlows of Morvan's disease are peculiar manifestations of syringomyelia. Changes in the hair, nails, and skin are common in neuritis and herpes zoster. Facial hemiatrophy presents a combination of trophic changes affecting all of the tissues involved.

## DEGENERATION

**The Stigmata of Degeneration.**—Walton has suggested that those signs upon which Lombroso laid so much emphasis as indicating such a departure from the normal as to constitute degeneracy are better spoken of simply as *deviations*, unless they indicate such a departure



FIG. 16.—IMBECILITY FROM DEFECTIVE PARENTAGE. Age 20. One of five defective children. (Dr. H. Work, Woodcroft, Pueblo, Colo.)

as to amount to an actual degeneration. All individuals possess some anatomical, physiological or psychical peculiarities, varying therefore more or less from the average normal standard, but unless the variation constitutes such a departure as to render the possessor in some manner or degree unfit for some of the relations of life, the term degeneracy should not be applied. There can be no doubt that the Italian school has over-shot the mark in attributing too much importance to minor deviations. An individual may possess several of the minor forms of anatomical stigmata, such as

flecks on the iris, high palatal arch, polydactyly, or webbed fingers or toes, without in any degree raising a presumption against his being of essentially normal type. A combination of several striking departures from the normal, such as albinism, microcephalus, and prognathism, should attract attention, since the possessor is likely to prove a well-marked degenerate, with epilepsy, sexual perversion, idiocy, etc., as possibilities. The progeny of degenerates is so notoriously likely to inherit the defects of the parents that society

must some day take more efficient steps for protecting its stream against such a pollution.

The anatomical anomalies of most importance consist in great departures from the normal in size, shape, and symmetry of the head, in facial asymmetry, deformities and malpositions of the ears, changes in shape and size of the jaws, microphthalmus, marked muscular insufficiencies, deformities of the hard palate, albinism, marked irregularities or imperfections in the teeth, failure or de-



FIG. 16A.—MONGOLIAN IDIOT. (Case of Dr. J. W. Ames, Children's Hospital, Denver.)



FIG. 17.—IMBECILITY WITH EPILEPSY. Age 14. Cerebrospinal meningitis at 2 years. Convulsions since that time.

fective development of the limbs, dwarfism, feminism in men and masculinism in women, and marked changes in development of hair or skin.

Physiologically, we have as notable departures, left-handedness, which is vastly more common in degenerates, but not to be regarded excepting in association with more serious deviations, slowness in learning to walk and talk, epilepsy, spasms, tics, nystagmus, deaf-mutism, daltonism, impotence, sterility, uncontrollable appetites, as for liquor

or drugs, and lack of the ability to endure the usual emotional and nervous strains of life.

Psychically, we have the various grades of feeble-mindedness, idiocy and insanity. Short of these are absence of self control, precocity, moral delinquency, sexual perversion, exaggerated egotism, etc. Excessive self-consciousness, and marked eccentricity signify little, unless in association with graver defects.

Degenerates differ so greatly in degree that they may be classified as:

(1) **SUPERIOR DEGENERATES.**—In these certain degenerative symptoms, perhaps of moderate degree, are present, but associated with brilliant qualities, generally in limited lines, often raising the individual to the rank of a genius. Many noted artists, musicians, mathematicians, etc., have belonged to this class. So long as the stigmata of degeneration are chiefly anatomical, the possessors may be mentally and physically sound.

(2) **INFERIOR DEGENERATES.**—These have the marks of degeneracy with such mental aberrations as to render them unfit for the ordinary associations of life.

(3) **DEBILES, IDIOTS, IMBECILES.**—Feeble-minded and others of the lowest class belong under this heading.

## SECTION IX

### EXPLORATORY PUNCTURES AND EXAMINATION OF THE FLUIDS OBTAINED

A HOLLOW needle is used for the purpose of exploring suspected areas, and especially to determine if fluid be present. A larger and longer needle than that ordinarily used on the hypodermic syringe may be employed with this instrument, or preferably a somewhat larger, easily sterilized syringe. It is needless to comment on the necessity of having the instrument in perfect order, or upon the need of surgical asepsis in using the needle.

With a needle of two or three inches (5 to 7 cm.) in length, and not over one-twelfth to one-twentieth inch in diameter (1 to 2 m.m.), there is little danger in an exploration, if done with reasonable judgment. I have known the puncture in a leukemic liver, explored upon the suspicion that it contained an abscess, to continue to bleed into the abdomen until death resulted, but this was rather from defective coagulability of the blood than the extent of the trauma. The spleen may bleed in similar manner if punctured when enlarged. In general, however, the heart and lungs cause but little trouble when the needle accidentally touches them, and the results to be attained are so important as to justify the risk we take.

The chlorid of ethyl spray, the application of pure phenol, or injection of one of the local anesthetic solutions may be resorted to if necessary. The needle should be pushed steadily forward, avoiding too sudden a movement for fear of disaster if a rib or other bone be encountered, and the piston is then withdrawn. If no fluid appear the needle should be slowly withdrawn, since it may have passed through a thin layer of fluid, which we might miss if the withdrawal were too precipitate. Before complete withdrawal, the direction may be changed and a new territory explored. If serious doubt exist, it is well to make a microscopical examination of any contents in the

eye of the needle, though we should rarely fail to find macroscopic evidence of pus if present. If one be fairly confident of fluid several explorations may be made. I have found pus in a small empyema upon the thirteenth puncture in one case, the entire region explored being little larger than the palm of the hand. The patient had been repeatedly explored by other physicians before without success, but his symptoms made it almost absolutely certain that pus was present.

Any movements imparted to the needle by respiratory or cardiac movements, etc., should be carefully noted. If we employ a needle connected by rubber tubing with a bottle and provided with a glass window, we may determine whether a cavity near the line of the diaphragm is actually above or below that line by the aspirating effect of inspiration upon the fluid in the tube, if it be in the chest cavity, or the expulsive effect if below the diaphragm. Solid tissue may occasionally be withdrawn in the hollow of the needle, especially if the needle has been rotated and pushed in and drawn out with the express idea of retaining the tissue encountered.

The fluid obtained should be first examined macroscopically. We may determine in many cases the presence of fibrin, blood or pus, and get some further ideas of its character by its odor. The latter may be that of an abscess with the characteristic colon bacillus infection, or may be urinous. With the microscope we may find blood cells, pus cells, bacteria, echinococcus hooklets, amebae, chylous fat droplets, ray fungi, etc.

The presence of a fair number of polymorphonuclear cells in an apparently serous pleural effusion should lead us to explore the lower part of the chest, since the pus cells often settle and leave an almost clear supernatant fluid.

The presence of food particles in fluid may signify a perforation of the stomach or other portion of the digestive tract. Cells or fragments from a tumor may be secured, cholesterin crystals may be found in old serous exudates, and hematoidin crystals in old purulent foci.

The specific gravity of the fluid, if in sufficient amount for the

procedure, may be taken, and then the chemical tests applied for albumin, sugar, urea, and mucin.

### REGIONS TO BE EXPLORED

The pleural cavity is the one most frequently calling for exploration. The traditional directions to aspirate in the seventh space in the axillary line or the sixth near the angle of the scapula are utterly insufficient. The exploration should be made at the center of the area of greatest flatness or dullness, wherever this may be, paying due regard to the position of the heart and great vessels. If the pleural cavity be full, the ordinary direction would suffice to find the fluid. On the other hand, an interlobar pleurisy or empyema, an abscess of lung, or an abscess of the liver pushing the diaphragm up sharply, might demand exploration, but would require some especial selection of the point of puncture to give any reliable results.

The pericardial cavity may be explored a finger breadth to the left of the sternum, in the fourth space, or slightly further to the left in the fifth, in the left costoxiphoid angle, passing the needle upward, or to the left of the apex-beat, or in some cases to the right of the sternum in the fourth space (when the effusion gives dullness far to the right). Great care should be used not to injure the heart and especially so since a thin-walled auricle, a coronary artery or Kرونеккер's coördination center might be injured. In general, exploration of the pericardial cavity should not be undertaken without a very definite need and after a most careful examination and study. I have seen blood aspirated from a much dilated heart with benefit, but not without danger.

The peritoneal cavity is commonly aspirated in the center line below the navel, after making sure that the bladder is empty. If no fluid be obtained the patient may be placed upon the side toward which the fluid gravitates, most readily and the puncture made between the navel and the anterior superior spine of the ilium.

The liver is often explored for abscess or hydatid cyst. If the indications are clear, as when a large cyst or abscess gives a rounded



area of dulness, perhaps bulging, the aspiration may be made in the center of this region, otherwise the needle may be entered in the center of the hepatic dulness, either in the anterior or mid-axillary line, or in the back. If it becomes necessary to search for an abscess of the liver not readily located, it is best to use a general anesthetic. In several cases I have had the abdomen opened and the liver exposed before using the needle, since the condition is serious enough to demand any necessary means of relief.

The gall-bladder should probably never be aspirated until it is exposed by the surgeon.

The spinal cavity may be entered at the region of the third, fourth, or fifth lumbar vertebra, the needle being entered one or one and one-half c.m. to the right or left of the median line, and passed somewhat inward and upward for 2 to 4 c.m. in children, or nearly twice as far in adults. The intervertebral spaces are widened and the operation is made more easy by bending the patient's body forward. The fluid escapes slowly drop by drop if pressure be normal, and faster, even in a stream when the pressure is increased, as in meningitis or brain tumor. There is commonly nothing to be gained by manometric measurement of the pressure to justify the additional time required.

Puncture of the spleen is occasionally performed to determine the presence or absence of a cyst or abscess. It is probably not justifiable to obtain blood for examination in typhoid or malaria, and may be dangerous also in leukemia.

Aspiration of the tumor in appendicitis is mentioned in some works, but the procedure, like that upon the gall-bladder, is antiquated, unsafe and useless.

Cysts, abscesses, etc., are to be explored with the needle only after careful consideration of their character, of the danger of infecting neighboring serous cavities with their contents, and of their relationship to important organs, especially the heart and large vessels. In general, they should be punctured at the center of the greatest area of flatness, and with a fine needle only, since the escape of fluid into the neighboring cavities may do harm.

The exploration of the lung for echinococcus cyst should be done

only after preparations have been made to open the chest in case of trouble; for death, virtually by drowning, has occurred through neglect of this rule. In attempting to demonstrate a lung cavity we should explore when the presence of flatness and the long abstinence from cough and expectoration lead us to believe that the cavity is filled. I have obtained pus from a bronchiectasis which led me to diagnose lung abscess, and even with the greatest care we may be deceived. Careful microscopic examination for epithelial cells, pus cells, elastic fibers, bacteria, etc., should be made. The presence of elastic fibers practically excludes an empyema as the source of the fluid.

The harpoon is occasionally used to obtain muscular tissue for examination for trichinae.

### EXAMINATION OF THE FLUIDS OBTAINED

**Specific Gravity.**—Ruess states that the following figures represent the relation between the specific gravity and the proteid content (in serous fluids):

Sp. gr. 1.018 higher than	4 %	proteid contents
Sp. gr. 1.015 lower than	2.5%	proteid contents
Sp. gr. 1.012 lower than	1.5–2 %	proteid contents
Sp. gr. 1.010 lower than	1 –1.5%	proteid contents
Sp. gr. 1.008 lower than	0.5–1 %	proteid contents

In general exudates contain much more protein than transudates.

In inflammatory exudates Runeberg states that the proteid content reaches 4 per cent. to 6 per cent. (including carcinoma and tuberculosis of the serous membranes). In congestive transudates he finds 1 per cent. to 3 per cent. and in pure hydremic transudates less than 0.5 per cent.

A specific gravity of 1.018 or over, indicating a proteid content of 4 per cent. or over and often accompanied with the formation of a distinct clot, indicates definitely an inflammatory exudate. Thus an accurate urinometer may give much assistance in deciding

upon the character of a given fluid. Transudates may contain blood or chyle in considerable amount.

For methods for the detection of biliary pigment, indicating a communication between the gall passages and the cavity from which the fluid has been obtained; of succinic acid in echinococcus fluid; of urea in cysts of the urinary system; of paralbumin in ovarian cysts, and pancreatic ferments in pancreatic cysts, the reader is referred to the larger works upon clinical diagnosis.

The presence of gas as demonstrated by the puncture may indicate an ordinary pneumothorax, puncture of the lung by the needle, or the hollow organs within the abdomen, perforation into the peritoneal cavity, pleura or pericardium by some ulcerative or traumatic process affecting the digestive tract, or the development of gas through action of bacteria, as notably in the urine of diabetics infected with the colon bacillus.

**Cytodiagnosis.**—The sediment obtained from aspirated fluids may be examined directly, or stained specimens may be prepared. In this manner the predominance of certain cells or their absolute number may be determined. If the fluid tends to coagulate too quickly for the examination it may be aspirated directly into a syringe partly filled with sodium citrate or other solution used to prevent coagulation.

**Red Cells, Leukocytes, Etc.**—We next examine the specimen for red cells, different varieties of leukocytes, epithelial, endothelial or tumor cells. Various bacteria may also be detected.

Red cells may be detected when the demonstration of blood by the macroscopic inspection is impossible, and even when chemical detection is more or less uncertain. The presence of blood in an exudate suggests first the possibility of a tuberculous or cancerous process, but it may be present also in the hemorrhagic diathesis, in exudates arising from marked venous congestive processes, and occasionally in those associated with disease of the kidney.

Because of the tendency of the heavy cellular elements to settle in the fluid, we must beware of basing any opinion upon the cells found in an aspirated fluid, without a consideration of the possibility that we may be dealing with the relatively clear supernatant fluid.

I have myself diagnosed a serous pleurisy upon seeing the clear fluid flow from the needle when I had confidently expected pus, to learn shortly that the subsequently obtained fluid contained myriads of pus cells.

An abundance of leukocytes signifies an acute infectious process, and is especially associated with infection by streptococci, staphylococci, pneumococci, meningococci, or colon or typhoid bacilli. In tuberculous pleurisy we generally find an increased proportion or even a great predominance of lymphocytes in the serous exudate. It is not rare, however, in early exploration to find polynuclear leukocytes in predominance, with many endothelial cells. Koniger states that after the second week in these cases the lymphocytes gain the ascendancy. There is a tendency for the polynuclear cells in a tuberculous exudate to degenerate quickly, shrivelling and breaking up. Confirmation by the finding of tubercle bacilli or by animal injection is always to be desired.

The French speak of "pleural eosinophilia," the proportion even reaching seventy-five per cent. regardless of the cytology of the blood. This finding is not at all pathognomonic, since it may be present in such varied conditions as tuberculosis, rheumatism, gangrene of lung, cancer, syphilis, etc.

In general we may say that lymphocytosis is significant of a mild irritation as the cause of the exudate, while polynucleosis signifies a severer irritation. The replacement of the former by the latter is suggestive of some complication, as, for example, that a serous pleurisy is about to become an empyema. Lymphocytosis is most commonly seen in effusion due to tuberculosis, syphilis, certain malignant tumors, and occasionally in uremia.

Occasionally cells detached from malignant growths are found in an exudate, and a positive diagnosis may be made from them under favorable circumstances. Much caution should be exercised.

In empyema attention should be given to the state of preservation or otherwise of the polynuclear cells. In an empyema associated with pulmonary tuberculosis or following a pneumothorax in this disease, granular and fatty transformation of the leukocytes is often complete, so that only nuclei and detritus may be recognizable.

In an ordinary infectious type of empyema, on the other hand, we find well-preserved polynuclear cells or at most a simple swelling with less affinity for stains. It is notorious that the infective agent is much more difficult to demonstrate in the tuberculous effusion than in the usual infectious ones, ordinary staining and cultural methods sufficing for the latter while inoculative experiments may even fail in the former. This may be due to the fact that even 20 or 30 c.c. of exudate may be necessary for an infection in the case of a guinea pig, and the animal may well succumb to the toxemia before the tuberculosis has time to develop.

**Inoscopy.**—By the digestion of the clot forming in a suspected tuberculous exudate by a solution of pepsin and the use of the centrifuge tubercle bacilli may often be obtained from the resulting fluid. They are likely to be shorter and broader than the organisms found in the sputum, and may be paler in color after staining.

**Cerebrospinal Fluid.**—The fluid obtained by lumbar puncture in health, a few cubic centimeters in amount, is clear, of a specific gravity of 1.003 to 1.007, and contains only occasional endothelial cells and leukocytes when examined under the microscope. The pressure of this fluid as obtained by a water manometer in the horizontal position of the patient is 60 to 100 m.m. but may rise to 200 or even 800 in meningitis or tumor of the brain. If the pressure falls to 60 m.m. the withdrawal should cease.

In disease even 100 c.c. may be withdrawn. In meningitis, acute hydrocephalus, brain tumor, certain infectious diseases and paresis, an increase is commonly found. Even though the intracranial pressure be greatly increased, as in hydrocephalus, there may be no excess of fluid in the spinal canal, owing to the closure of the communication between the canal and the basilar spaces of the brain.

The fluid is likely to be clear and colorless in brain tumor, hydrocephalus or even in tuberculous meningitis, while in acute meningitis, especially of epidemic form, the fluid is yellowish and even creamy in appearance. The fluid may be stained yellow but remain clear in jaundice. Blood may color it in hemorrhage into the ventricles, and the trauma of the needle may at times cause such a discoloration.

**Cytological Examination.**—In the spinal fluid of tuberculous meningitis lymphocytes predominate, and also in syphilis affecting the nervous system and other chronic processes. In the acute infections, such as are caused by the meningococcus, staphylococcus, streptococcus, typhoid bacillus, colon bacillus and pneumococcus, a polynucleosis is noted.

In addition to the organisms mentioned the influenza bacillus, bacillus of glanders and the *Bacillus pyogenes furtidus* are occasionally found in the spinal fluid. The trypanosomes of the African sleeping sickness should be mentioned. Streptococci may be present with them.

Noguchi's butyric acid test for syphilis may be applied to the cerebrospinal fluid, and is claimed to be positive in a greater per cent. of cases than the Wassermann reaction or the cytodiagnostic test. It is still under investigation.

In those cases of tumor of the post-cranial fossa or other disease blocking the communication between brain and spinal canal, puncture of the lateral ventricle is occasionally performed and often followed by a decompression operation if the pressure be excessive. For details as to exploratory puncture of the brain the reader is referred to the special treatises.

**Chylous Fluids.**—These are turbid from the presence of fine particles of fat. This may reach a proportion of  $\frac{1}{2}$  per cent. to 2 or 3 per cent. Its solubility in ether aids in its detection. The appearance of the fat droplets under the microscope is characteristic. In the pseudochylous exudates no free fat can be detected, yet the opalescence seems the same as in the true forms. Degenerated epithelial or endothelial cells, bacteria, lecithin, various globulins, etc., have been suggested as the cause of the turbidity in different cases. The typical chylous effusion is abdominal and due to interference by pressure with the circulation in the thoracic duct or lymphatic vessels. The pleural cavity and the pericardium are less often involved.

**Pancreatic Cysts.**—The fluid escaping from a pancreatic fistula commonly digests the skin in a very characteristic manner, and suggests the attempt to test its ability to digest albumin in an alkaline

medium. In the fluid of an old pancreatic cyst the test may fail without at all throwing doubt upon its pancreatic origin.

**Hydronephrosis.**—In the chronic cases with occlusion of the ureter the function of the kidney may be suspended so that no characteristic urinous features may be noted in the fluid. The presence of urea and uric acid, with tubular and cellular elements in the sediment, ordinarily makes the diagnosis easy, though the two former substances have rarely been found in ovarian and pancreatic cysts.

**Hydatid Fluid.**—This normally is as clear as water, contains considerable sodium chlorid, and the presence of daughter cysts, cyst membranes, scolices or hooklets, is easily determined. Granular cells, cholesterin crystals and detritus may also be found with the microscope. In suppuration of the cyst the qualities of a purulent fluid are superadded.

**Ovarian Cystic Fluid.**—This varies enormously in appearance, consistency and composition. It may be watery, viscid, jelly like, yellow, reddish or brownish. The specific gravity may vary from 1.010 to 1.040, and a great variety of epithelial cells, blood cells, fat globules, fatty crystals, etc., may be noted.

Dermoid cysts contain a fatty material derived from the epidermal lining, with desquamated epithelial cells, and, macroscopically, hair, teeth, bone, etc.

In the fluid from a spermatocele we may find leukocytes, spermatozoa, fat granules and detritus. The fluid is ordinarily alkaline but may be acid, and does not coagulate. From a hydrocele a coagulable fluid is obtained with a specific gravity of 1.015 to 1.030 and with considerable albumen, and large oval nucleated cells, perhaps grouped in masses, are found. The presence of many lymphocytes points to a tuberculous origin of the fluid.

## SECTION X

### EXAMINATION OF THE BLOOD

We are able to give space only for the usual diagnostic procedures of hematology and refer to the larger and special works for fuller details.

The blood may be drawn preferably from the cleansed and dry tip of the least used finger or from the ear if desired, by means of a special instrument, or an ordinary surgical needle, the latter being easily obtained, thoroughly efficient, easily sterilized and less portentous in appearance than anything else used for the purpose. For cultural and other complicated procedures the use of a needle inserted into a vein is necessary.

The determination of the specific gravity of the blood, of its freezing point and the degree of its alkaline reaction, etc., may well be left to the laboratory worker being of slight clinical importance.

### COAGULATION TIME

This is of importance in the diagnosis and therapy of certain diseases. Sahli gives as the best method the following: A drop of blood and one of distilled water are placed together in the hollow of a ground slide and kept at a temperature of 25° Centigrade. By touching the fluid lightly every half minute with a fine glass rod the appearance of the first threads of fibrin is determined.

The time of coagulation is normally from 2 to 8 minutes, generally 5 or 6, and a time beyond 9 minutes is abnormal. In the purpuras it may extend to 10 to 20 minutes, in hemophilia even 50 minutes. In jaundice, hemoglobinuria, dropsy and asphyxia it is increased, as after the bite of the cobra and certain other poisonous snakes. The coagulation time is decreased in stasis, after transfusion and hemorrhage, in hunger, after the use of calcium chlorid and



in many acute diseases. The actual amount of fibrin is increased in pneumonia, articular rheumatism, and certain other acute diseases. It is decreased (hypinosis) in pernicious anemia, leukemia, purpura, malignant disease and other wasting diseases.

### HEMOGLOBIN

The blood contains about 14 per cent. of hemoglobin but we express its amount clinically in the percentage of the normal. This percentage varies normally somewhat at different ages, being lower in childhood. It is increased in high altitudes. We may obtain some idea of the percentage of hemoglobin by inspection of the blood drop, especially if it be allowed to fall on a dull white absorbent surface.

Tallqvist's chromolithographic scale, in which the colors correspond to those of certain hemoglobin values as shown by the drop of blood upon a piece of white filter paper is the most commonly used clinical test for the amount of hemoglobin. Although inexact and capable of giving but a rough estimate at best, its simplicity and time-saving features are of the utmost importance to the clinician. What he seeks to know is whether the hemoglobin varies materially from the normal and not the exact degree of the variation, which is of no especial value to him. The comparison of the drop of blood and the scale should be made at once, before the action of the air and possible drying can influence the color.

Von Fleischel's hemoglobinometer is the one most commonly used. Under a stage with a central opening is fitted a short glass-bottomed cylinder, divided vertically by a partition. Beneath one compartment is arranged a wedge-shaped piece of colored glass movable by means of a screw, and furnished with a scale. A white reflector is placed under the stage. One compartment is filled with distilled water, that in the other being colored to a certain degree by the admixture of blood, measured by means of a certain pipette accompanying the instrument, and obtained from the patient under examination. The tint of the colored glass under the water-containing compartment is brought to match that of the blood-containing

compartment, when the percentage of hemoglobin may be read from the scale.

Many other methods for the estimation of hemoglobin have been introduced but the two mentioned suffice for clinical work.

The percentage of hemoglobin may be estimated from the specific gravity if this be available. Schmatz finds that the specific gravity of 1.030 corresponds to 20 per cent. hemoglobin; 1.035 to 30 per cent., 1.038 to 35 per cent., 1.041 to 40 per cent., 1.0425 to 45 per cent., 1.0455 to 50 per cent., 1.048 to 55 per cent., 1.049 to 60 per cent., 1.051 to 65 per cent., 1.052 to 70 per cent., 1.0535 to 75 per cent., 1.056 to 80 per cent., 1.0575 to 90 per cent., 1.059 100 per cent.

Much interesting information is obtainable by the examination of the blood by the spectroscope, but this does not come within our range.

Oligochromemia is observed in secondary anemias, pernicious anemia, leukemia, chlorosis, in chronic infections, chronic nephritis, in the cachexia of malignant disease, mineral poisoning, etc. If the hemoglobin be below 40 per cent. the utmost circumspection should be used before undertaking any surgical operation, although permissible in certain cases.

**Color Index.**—This term expresses the relationship between the amount of hemoglobin present in a given case and the amount which theoretically should be present with the same red blood count. It is the quotient obtained by dividing the hemoglobin percentage by the percentage of red blood cells. The color index is normally 1.0, expressive of a value of 100 per cent. of hemoglobin associated with five million red blood cells. If both hemoglobin and red blood cells diminish in the same proportion it remains unchanged. If the hemoglobin be reduced more markedly than the red blood cells the value is less than 1.0 as in chlorosis and splenic anemia. In pernicious anemia on the other hand, the cells are reduced more in proportion than the hemoglobin and the index rises even as high as 1.9. We must beware of basing judgment upon this factor in the diagnosis alone, yet it is of much value if taken into consideration with all the rest of the evidence.

### THE BLOOD COUNT

The estimate of the number of cells in the blood depends upon an exact dilution of the blood and the count of the cells in a measured quantity of the solution.

**Thoma-Zeiss Apparatus.**—The Thoma-Zeiss counting apparatus is almost universally used in this country. The apparatus is fully described in the larger works. For the red cells, the blood flowing from the needle puncture in the finger tip (and not expressed) is sucked up into the appropriate tube to the mark 1 and the ampulla filled to the mark 101 with Hayem's fluid. After thorough shaking we have a uniform mixture of 1 part to 100 of blood. After expelling a few drops, a drop is placed in the counting chamber over the ruled scale, and the cover adjusted. The counting is done under a medium power (one sixth or lower) cells touching the upper and left boundaries of any square being counted as belonging to that square, to obviate any doubt as to which have been counted, those touching the opposite sides being neglected. For an accurate count a large number of horizontal rows of 20 squares each should be counted, but a reasonable approximation may be obtained by counting as a unit four squares in a row, taking the average number of cells in a considerable number of such counts, and multiplying by 100000. (One small square equals  $1/10$  depth by  $1/400$  area, making  $1/4000$ . Hence four squares taken together equal  $4/4000$  or  $1/1000$ . The blood has been diluted 100 times, hence the factor is 100,000).

For the more exact count 400 squares should be utilized. In this case we take the size of the small square as  $1/400$  m.m. x  $1/10$  m.m., its depth, equal to  $1/4000$  m.m. Since the dilution is  $1/100$  the factor becomes 400,000. By multiplying the average number of red blood cells in the small square by this factor we obtain the total number. If 400 squares contain 4,000 cells the number per square is  $10 \times 400,000$  equals 4,000,000, the number of red cells per cubic millimeter.

If the count of red and white cells is to be made at the same time

Toison's fluid is advisable, since it colors the leukocytes, and renders them easily distinguishable.

**Leukocytes.**—With the white pipette the (large) drop of blood is drawn with gentle suction into the tube to the mark 1, and diluted ten times by the introduction of the  $\frac{1}{10}$  per cent. acetic acid solution, used to render the reds invisible. Methyl-green, methyl-violet or other coloring agent makes the count easier by rendering the nuclei of the leukocytes more easily visible.

The drop is placed upon the ruled disc, and all the leukocytes in all the small squares (400) are counted. If greater exactness is desired a greater number of separate drops are counted.

If we find, for example, that there are 80 white blood corpuscles in 400 squares the percentage is 0.2, and our factor in 40,000, the product of  $1/4000$ , area of one square, by  $1/10$ , depth of square. Thus  $40,000 \times .2$  gives 8,000 as the number of leukocytes per cubic millimeter. Many supplementary methods have been devised and may in fact easily be devised by the expert, to be used in case different apparatus be employed (as the red pipette for the white count) or in case the number of reds and whites varies greatly from the normal, as in severe anemia or leukemia. We have space but for a single standard method in each case.

**The Hematocrit.**—The volume of cells in the blood may be determined by the use of this instrument. The white cells are thrown to the extremity of the rapidly revolving tube, the red cells are superimposed and the plasma floats above. The instrument has not come into general use since the counting apparatus is much more accurate.

In general we may assume 5,000,000 red cells to be normal for men, and 4,500,000 for women. In Switzerland and other elevated countries these figures stand from one-half to one million higher. Dilution of the blood by eating and drinking, or concentration by starvation, cause respectively a decrease or an increase in the proportion. Obesity tends to lower the number.

The ratio of white cells to reds in adults is approximately 1 to 520, but with great variations even in conditions of health. The absolute number of leukocytes in health may be stated as 7,000,

but it varies from 5,000 to 10,000, and is normally greater for children.

### THE RED CELLS IN DISEASE

The smallest number of red cells per cubic millimeter is doubtless found in pernicious anemia, Quincke's count of 143,000 being rarely if ever surpassed. A count of 1,000,000 is common in this disease. In chlorosis and other milder anemias the count may be from 2 to 4 million. In general, oligocythemia indicates some type of anemia, and further examination is necessary to determine the exact form.

A less frequent condition is polycythemia. It is found in the newborn, after blood transfusion, often in very robust individuals, and in those living at considerable altitudes. Polycythemia may be present because of abnormal concentration of the blood, from diarrhea, profuse sweating, the abstraction of much fluid from one of the cavities of the body with resulting concentration of the blood from the reestablishment of the effusion, etc. In cyanosis the blood stream is often slow, and presents an increase in the number of red cells. In chronic cyanotic polycythemia, the count may rise to 9 to 11 million, a number unknown in any other condition. In one of my cases the number was just under 10 million. In a recent case of the so-called enterogenous cyanosis marked polycythemia was present, as in other reported cases.

**Abnormal Erythrocytes.**—Red cells are normally  $7\frac{1}{2}$  microns in diameter. If a large number are found measuring 6 microns or less we speak of the condition as microcythemia (notable in chlorosis). If the cells exceed 9 microns they are called macrocytes (macrocythemia). Pernicious anemia shows an unusual number of large red cells.

Microcytosis is thought to be due to rapid and perhaps defective blood formation. Macrocytes are believed to be developed in the marrow.

Poikilocytes are irregularly shaped cells, oval, pear-shaped, elongated, and wholly irregularly shaped and commonly varying greatly in size. The degree of poikilocytosis depends roughly upon the

severity of the anemia, and the worst forms are noted in pernicious anemia.

These irregularly shaped cells vary greatly in hemoglobin content and staining capacity. They may be so pale as to be mere shadows or so dark as to stand out prominently in a group of paler cells.

Polychromatophilia is frequently found in pernicious anemia, the cells reacting indifferently and irregularly to basic and acid dyes. The so-called "stippling" or granular basophilia noted in severe anemias and leukemias, but most characteristically in lead poisoning, is due to the presence of granular areas in the cytoplasm, with an affinity for basic stains. The so-called "ring-like bodies" have no especial significance so far as known.

In the stained film we may find nucleated red cells. Normoblasts are otherwise normal red cells containing a nucleus. They are thought to be probably young cells. The nucleus stains a deep bluish color with the Ehrlich and other stains. Normoblasts are found normally in the blood of infants of a few days, and in the bone marrow and spleen of adults.

Megaloblasts or giantoblasts vary from 11 to 20 microns in diameter, often slightly oval or irregular in shape, with a large nucleus staining less deeply than the nucleus of the normoblast. The cytoplasm often stains atypically. Megaloblasts are found normally only in the fetal bone marrow. In pernicious anemia and in leukemia their abundance in the blood is of grave omen. A few in the blood of a simple anemia are of no great significance. They are commonly associated with normoblasts in these cases.

**Blood Platelets.**—These are small spherical or irregular bodies from 1 to 4 microns in diameter and pale yellowish in color. They disintegrate upon exposure to the air and have no ameboid activity. Wright believes that they represent fragments of giant marrow cells; 200,000 to 300,000 per c.mm. are commonly present. The number is lessened in pernicious anemia and often increased in secondary anemia, leukemia, rheumatoid arthritis, etc. We know but little of their significance.

Lipemia and melanemia are of little interest clinically.

## LEUCOCYTES

The polymorphonuclear cells are commonly present in a proportion of 60 per cent. to 75 per cent. (about 5,000 per c.mm.); the small lymphocytes 20 per cent. to 30 per cent. (1,500 per c.mm.); large lymphocytes 5 per cent. to 6 per cent. (400, more or less); eosinophiles 2 per cent. to 4 per cent. (100 to 200 per c.mm.); transitionals 1 per cent. (50 to 100). Considerable variation from the above may be noted in the figures given by different authorities, and the figures are thus to be taken *as approximate only*.

In the fresh preparation of blood the leukocytes appear as pale, colorless cells, generally slightly larger than the red cells, commonly having ameboid movement.

Polynuclear leukocytes (polymorphonuclear neutrophilic leukocytes) vary from 9 to 12 microns in diameter and are characterized by a polymorphous irregularly bent nucleus, often appearing as multiple nuclei, as when the cell is treated with dilute acetic acid. The nuclei take the usual stains freely. The neutrophilic granules are best shown by Ehrlich's stain, taking a violet color.

These cells arise from bone marrow. They show the ameboid and phagocytic properties better than the other cells. When recovered from pus they stain less typically than when obtained from the blood.

The small lymphocytes come next to the polynuclear cells in proportion. They are of the size of the red cells or slightly smaller, with a single round nucleus, staining deeply, with only a thin rim of protoplasm around it, homogeneous, and without granules. They are devoid of ameboid movement and are not phagocytic. They are derived from lymph glands and other lymphadenoid tissue, and possibly in part from the bone marrow.

The large mononuclear leukocytes have a diameter of 12 to 20 microns, and generally show a large eccentrically situated nucleus, which stains faintly. A distinction is made by many between the large lymphocyte proper, presumably originating in lymphatic tissue, and the large mononuclear cells supposed to come from the bone marrow.

Transitional cells seem to lie between the forms last described and the polynuclears, and present an indented or constricted nucleus. Neutrophilic granules appear in small numbers.

**Eosinophiles.**—These cells resemble the polynuclears, but the neutrophilic granules of the latter are replaced by coarser oxyphilic granules, showing red or orange color with Ehrlich's stain. The cells are developed in the bone marrow. Those found in the sputum in asthma and bronchitis are often mononuclear and are believed to be degeneration forms of the polymorphonuclear ones of the blood stream.

A few mast cells may be present in normal blood. They are of the polymorphonuclear or transitional type, with large, uneven and irregularly distributed basophilic granules, staining a deep blue, with alcoholic methylene blue or Jenner's stain. They arise from the bone marrow.

**Pathological Leukocytes.**—Myelocytes (better neutrophilic myelocytes). These are believed to be the progenitors of the neutrophilic cells and to remain normally in the bone marrow. They are often larger than the largest normal leukocytes, but may be little larger than the red cells, and are non-ameboid. The myelocyte has a large, faintly staining nucleus, like that of the large lymphocyte, but the cytoplasm contains many neutrophilic granules, showing especially well with the Ehrlich stain. They are characteristic of myelogenous leukemia, but are also found in malignant tumors involving the bone marrow, and in some cases of leukocytosis (infants').

Eosinophilic myelocytes are of much the same character as the last described cells, but have eosinophilic instead of neutrophilic granules. Ehrlich believes them to be the progenitors of the normal eosinophilic cells. They are found especially in myelogenous leukemia.

**Irritation Forms.**—Türk describes under this heading "mononuclear cells which resemble the lymphocytes in their characteristics, but are sometimes larger and without granules." The nucleus is relatively smaller. Leishman's solution stains the nucleus a deep violet and the protoplasm blue. These cells are found under the same conditions as the myelocytes, as a result of the irritation of the bone marrow.



Various atypical lymphocytes are found in severe anemias, and in lymphatic leukemia, and no two hematologists would exactly agree in their classification. Degenerated leukocytes are often found, even in normal blood, staining feebly and perhaps devoid of granules.

### MICROSCOPIC EXAMINATION OF THE BLOOD

A small drop of fresh blood is permitted to spread evenly between the clean cover glasses upon which it has been obtained, with the utmost care to avoid pressure or sliding movements. By smearing the edges of the cover glass with oil, drying out of the specimen may be postponed. It should be examined immediately. Poikilocytosis, anisochromia, the presence or absence of rouleaux formation, and of ameboid movement, the presence of malarial plasmodia, the spirilla of relapsing fever, the filaria sanguinis hominis, etc., may be noted. With training one may often form a good judgment as to the presence of a polynuclear leucocytosis by inspection of the slide. Myelogenous leukemia may be easily recognized.

In obtaining the blood films for staining it is best to place the cover glass which has been touched to the small drop of fresh blood upon the finger, upon another glass, allow the drop to spread by capillarity alone, separate the two by a sliding movement without pressure, permit the film to dry, and stain it at convenience. Many prefer to spread the blood drop upon one slide by pushing it along with the edge of another slide. The film is fixed either by heat (115° C. or more) for fifteen or twenty minutes, or better by use of a stain which first fixes and then stains. Leishman's stain is extremely satisfactory. A few drops are placed upon the dried film, allowed to stand 30 seconds, then diluted with successive drops of distilled water until a peculiar shimmer appears upon the stain (6 or 8 drops commonly suffice). After five minutes this is washed off, the smear dried and mounted in balsam. Wright's stain acts in a similar manner and is much used, like Leishman's, in place of Ehrlich's and others, which require a previous fixation of the smear.

The various stains are taken in different degree and selectively

by the nuclei, protoplasm, granules, etc., of the different cells, and render their differentiation easy.

In the red cells we may recognize anisocytosis, polychromatophilia, anisochromia, oligochromia, the stippling of the cells, mentioned elsewhere, and the presence of nuclei in the various forms of nucleated red cells. The white cells are differentiated as described elsewhere. The nucleated and other abnormal red cells may be counted in the same manner as the white cells (*see below*).

**Differential Count of Leukocytes.**—One thousand white cells are counted in the stained specimen, and classified in accordance with their characteristics. A lesser total number will often suffice. By the use of the mechanical stage the covering of the field accurately is rendered much easier and more certain.

### LEUCOCYTES IN DISEASE

**Leukocytosis.**—A moderate hyperleukocytosis often occurs in health, as in infants and pregnant women, after exercise, after eating, etc. The proportion of the different cells remains normal. A pre-agonal leukocytosis is often noted. In gastric cancer the normal increase after meals does not occur, but much care must be exercised in arriving at this conclusion.

**Polymorphonuclear Leukocytosis.**—This is the form commonly seen in disease and consists in an increase in the number of polynuclear cells, generally relatively and absolutely, in the blood. In most cases we find the count between ten and thirty thousand, but it may reach fifty or one hundred thousand. This type of leukocytosis occurs in acute disease when some focus of inflammation is present, as in abscess, pneumonia, tonsillitis, pleurisy, peritonitis, appendicitis, meningitis, pyemia, etc. If the disease gives rise to no local reaction, as in typhoid and tuberculous meningitis, there may be no increase in the polynuclear cells. In tuberculosis of the lungs a leukocytosis may occur only after secondary infection has taken place in the cavities formed by the tubercle bacillus.

An increase in the number of polynuclear cells is often found in the cachexia of malignant disease, especially if the growth is a rapid one, or if metastases have occurred. After severe hemorrhage a simi-

lar increase is often noted and after the use of ether, chloroform, quinin, potassium chlorate, and certain other drugs.

In general we may say that the modern tendency is to place less dependence upon the white blood count in diagnosis than formerly, and especially to consider it only in relation to the other signs and symptoms of the case. Leukocytosis is so dependent upon the relationship existing between the infection and the body resistance, and varies so greatly in cases which may appear quite similar, that great caution must be exercised in estimating the value of the count.

**Lymphocytosis.**—The child has a normal increase in the proportion of lymphocytes as compared with the adult. An absolute lymphocytosis is fairly characteristic of lymphatic leukemia and malignant disease affecting the bone marrow. A relative lymphocytosis may be noted in syphilis, measles, pertussis, scarlet fever, typhoid, pernicious anemia, Hodgkin's disease, rickets, and various other conditions, but is of much less diagnostic value than the absolute form mentioned above. Cabot states that rarely a lymphocytosis may occur under just the conditions that would lead us to expect a polynucleosis.

**Eosinophilia.**—An enormous increase in the proportion and the absolute number of eosinophiles is to be found in trichiniasis, and may be almost diagnostic of the disease. The number may exceed that of all the other leukocytes combined. I have also seen it in less degree in ankylostomiasis and echinococcus cyst disease, and it may be found in almost any intestinal helminthiasis and in Bilharzial infection. In bronchial asthma, occasionally in syphilis, in pemphigus and certain other skin diseases, and in neurasthenia, it is often noted.

Myelocytosis signifies the presence of large numbers of the abnormal white cells characteristic of myelogenous leukemia. A few myelocytes may be found in pernicious anemia and the various secondary anemias.

Basophilia is rare and of little clinical significance.

**Leukopenia.**—A decrease in the number of leukocytes is noted in starvation and malnutrition. More important is the decrease noted in certain diseases, especially when contrasted with the increase in others above mentioned. A low count is common and more or less char-

acteristic in typhoid, tuberculosis, malaria, measles, German measles, and influenza. In many anemias leukopenia is found.

**Bacteremia.**—The presence of bacteria in the blood may occasionally be demonstrated in the stained slide, but cultural methods have displaced this process, excepting for certain organisms, notably the bacillus of anthrax.

Bacteria are generally few in number in the blood, but with proper technique many different forms have been demonstrated. Their presence is to be taken as absolute evidence of disease.

The pus cocci may be demonstrated in most cases of sepsis. In 80 per cent. of the cases of typhoid fever the Eberth bacillus has been proven. The organisms of pneumonia, tuberculosis, epidemic meningitis, plague, glanders, Malta fever, gonorrhea, influenza, leprosy, and other diseases have been isolated by cultural methods.

The spirochetes of relapsing fever are easily found in the febrile period of the disease, and the *Treponema pallidum* has been found in the blood in secondary syphilis, and even in hereditary syphilis. The *Trypanosoma gambiense* is found in the blood in African sleeping sickness. Herrick and Janeway have found the embryos of the trichina in two cases and others have been reported. The filaria embryos, especially of the nocturnal variety, are of extreme importance in diagnosis. Progress is so rapid in this general branch of study that we expect a great number of additions to the varieties of living organisms detected in the blood in the near future.

The plasmodia of malaria are best sought in the fresh specimen of blood thinly spread and examined at once by means of the immersion lens. The pigmented forms are most easily found, the flagellate types and crescents with greater difficulty, while the hyaline forms are very uncertain of detection. Since only one organism may be present in several fields, a lengthy search may be necessary for the demonstration of the parasite, or even a longer one, before one can reasonably give a negative answer. The fresh blood is best examined at the bedside, and should preferably be taken within a few hours of the time of occurrence of a paroxysm. The administration of quinin may utterly defeat the procedure.

In the red blood cells one may find the actively moving dark

pigment granules of the living plasmodium. The moving flagella attract attention by the motion communicated to the adjacent cells. Large red cells swollen by the growing parasite may be seen, or pale cells containing the clear hyaline forms with ameboid motion. Pigmented phagocytic leukocytes may be detected, the pigmented fragments of destroyed cells and organisms having been taken up. The hyaline bodies are easily confused with the more numerous vacuoles, often seen as artefacts in the red cells. If the beginner should find too many supposed parasites of this form he should suspect such an error.

A more certain method, under most circumstances, of finding the malarial parasites is the examination of the film, stained with borax methylene blue or Jenner's, Romanowsky's, or other stain, according to the taste and experience of the examiner. Staining is of especial service in studying the tropical forms of parasite because of the small amount of pigment commonly present.

In addition to the finding of the plasmodia, one may note the marked anemia commonly resulting from malaria, with leukopenia. A marked leukocytosis is occasionally found, however, in severe estivo-autumnal attacks. The presence of granules of free pigment or of pigmented leukocytes is of some value in diagnosis if the parasite be not found. For the differentiation of the different varieties and the study of the sexual cycles of development we must refer to special treatises. Thayer and Hewetson, many Italian, German, and English authors, and our own tropical surgeons have furnished abundant material for study in this line.

We shall also refer the reader to the special works for the description of the Widal test, the Wassermann reaction, the taking of blood cultures, and the performance of Bremer's test for diabetes.

## PART II

### ON THE CLINICAL APPLICATION OF DIAGNOSTIC METHODS

We have in the first part of this volume studied individual symptoms and diagnostic methods in a general way, with their details and the manner of their application. We shall now take up the other end of diagnosis—namely, the application of our methods already studied to the direct diagnosis of individual diseases, and with especial reference to differential diagnosis.



## SECTION I

### SPECIFIC INFECTIOUS DISEASES

#### A. BACTERIAL DISEASES

##### I. TYPHOID FEVER

**Definition.**—This is a continued fever, the result of infection by the *B. typhosus*, characterized by tenderness in the abdomen, diarrhea, enlarged spleen, a scanty eruption over the abdomen, great loss of flesh and strength and a greater number and variety of complications than any other disease. Its anatomical basis consists in the hyperplasia and eventual ulceration of Peyer's patches in the small intestine.

It prevails especially in the autumn months, and is the common continued fever of the temperate zone. All civilized persons are probably subjected to danger of infection sooner or later, and susceptible individuals contract the disease, generally in the second and third decades. One attack offers reasonable protection against a recurrence. The reason that it is relatively rare after middle age is presumably because persons at that time of life have had it or are insusceptible to infection excepting under extraordinarily favorable circumstances. It owes its prevalence throughout the civilized world to the one fact that sufficient care is not taken to prevent contamination of water and food supplies by human excrement, the infection of water by sewage being the one great element in the causation. Because of lack of facilities for prevention of contamination of water and food, the disease has always been a scourge of armies. Even if acute cases are not present upon assembling, the presence of bacillus carriers is almost certain in any large collection of men. Stone states that 4 per cent. of those recovering from typhoid become chronic carriers of the infection.

The bacillus of Eberth is constantly present in typhoid fever,



being excreted in the stools, and frequently in the urine. It may be detected in the blood and is present in the pus of many secondary infections (abscesses, necrosis of bone, pyelitis, etc.). It is carried not only in contaminated water and food but by the house fly, by oysters fed in contaminated waters, by dust, by vegetables eaten raw after application of sewage to the ground, etc. The great epidemics come from infection of a common water supply in most instances, and isolated groups of cases from some of the other causes mentioned.

The sexes are almost equally susceptible to the disease if exposed equally. Certain families possess a relative immunity, others an unusual susceptibility.

The period of incubation is generally thought to be 10 to 20 days, but probably varies with the intensity of the infection and the susceptibility of the individual exposed.

**Prodromata.**—Most constant are headache, exhaustion, malaise, bad taste in the mouth, nosebleed and irregular diarrhea. Chill or chilliness, cough and vertigo are less constant. The unusual cases of sudden onset often begin with a chill. The duration is commonly counted from the time of taking to bed, though this is variable according to the severity of the infection and the resistance of the patient.

The course is generally from three to four weeks but well recognized cases with positive Widal reaction have been recorded which had a normal temperature on the tenth day, and on the other hand the course may run six or seven weeks.

**Varieties of Typhoid.**—Such variations in severity and type of the disease exist that it is common to make a classification of the different forms. These depend upon the severity of the infection and the resistance of the individual, and especially of his different tissues, to the infection. Since the meninges, gall-passages, lungs or kidneys may bear the brunt of the attack, it is not strange that many cases vary much from the usual abdominal type. Complications often overshadow the original disease and add to the confusion.

(1) **ORDINARY FORM.**—This is the usual intestinal type with moderately severe infection and running a fairly typical course,

such as we describe elsewhere. The lesions are the typical ones of typhoid.

(2) MILD FORM.—This is the type with moderate fever, few complications, if any, and short course. It is still called “gastric fever,” “mountain fever,” etc., by those who do not avail themselves of the Widal reaction or other laboratory methods of diagnosis.

(3) AMBULANT FORM.—“Walking typhoid” is fairly common amongst men who habitually pay little attention to their feelings, less common in women. The patient suffers from headache, mild diarrhea, lack of appetite, sleeplessness and weakness, and may present himself in the consulting room with a temperature of  $102^{\circ}$  or  $103^{\circ}$ , a full crop of rose spots, and an enlarged spleen. Many of these patients spend their entire strength moving about before being finally sent to bed, and the mortality is unduly high on this account. A medical student whom I saw died a few hours after leaving the lecture room, of perforation, the diagnosis being established at autopsy.

(4) ABORTIVE FORM.—This variety often terminates suddenly by crisis, sometimes with sweating, during the second week, after showing a fair picture of typhoid during the first few days.

(5) GRAVE FORM.—In this type the fever, delirium, coma, tympanites, diarrhea, and other abdominal symptoms, carphologia, rapid pulse and, in general, all the severe symptoms of typhoid reach their maximum. The patient may die in the second week or resist the disease to finally fail even in the second month. The hemorrhagic septic form is the worst type of typhoid.

The rare afebrile form has been mentioned.

In children the disease is less common than in adults, though since the laboratory methods of diagnosis have been introduced it is found not very uncommonly in babies of the second and third years, and even in nurslings. It develops more quickly and often runs a shorter course. Rose spots and intestinal complications are less common, nervous symptoms are prominent and many of the cases are doubtless diagnosed and reported as meningitis. The remittent type of fever so often noted goes with the higher recovery rate in children. Convulsions occasionally initiate the attack.

Noma and gangrene of the vulva may be noted. The possibility of pyelitis or cystitis as a cause of the fever should be recognized. Many cases are diagnosed only by the application of the Widal reaction or other laboratory test.

In those past middle age the attack is less severe, as a rule and the eruption may be absent. The tendency to cardiac failure and respiratory complications offsets the favorable features.

Pregnant women commonly abort, but I have seen mother and child recover, when the abdomen at the time of labor was covered with rose spots and the temperature was  $104^{\circ}$  at the time of delivery. The maternal mortality is stated to be 15 per cent. to 20 per cent. and many of the children are lost, especially as premature labor is common. The fetal blood may react positively to the Widal test.

**PARATYPHOID FEVER.**—The failure to obtain a typical Widal reaction in certain cases of continued fever, thought to be typhoid, has led to the discovery that they are not due to the typhoid bacillus, but to certain organisms designated as paratyphoid (A and B) closely resembling the bacillus of Eberth, but differing in cultural and agglutinative properties. In some respects they approach the colon bacillus. Certain infections by these three organisms may be confounded with typhoid, but do not differ so markedly amongst themselves as different cases of true infection by the typhoid bacillus may do. The lesions in the autopsy room are not very characteristic of typhoid. Fortunately the treatment does not vary, and the prognosis is in general rather better than that of the true disease. Doubtless whole epidemics pass for typhoid when no bacterial studies are made. (*See Paratyphoid Fever.*)

**Symptoms.**—During the first week the temperature gradually rises, often averaging a degree F. a day, until it reaches  $104^{\circ}$  or  $105^{\circ}$  F. in the evening. In the cases of ordinary severity it holds this course for a week or more, with morning remissions of one or two degrees. The headache, thirst, delirium, flushed face, nosebleed and slight cough of the first week may continue, often with the addition of diarrhea in the second week. Often the advent of a well-marked typhoidal state prevents further complaint of headache, lassitude and thirst. The dichrotic pulse is more common in

the stage of onset than in any other disease. The white-coated tongue, often red at the tip and the edges, is likely to become more brownish, in part because of the mouth-breathing of the typhoid state. Although constipation is the rule in the first week, diarrhea is not uncommon, and it is more frequent in the second week. The spleen may now commonly be felt. The signs of mild bronchitis appear in the chest, the pulse shows a frequency of 90 to 100 or even 120, the abdomen is slightly full and tender, with gurgling if diarrhea be present. The rose spots appear over the abdomen and lower chest, but almost as constantly over the loins, on the sixth to the eighth day, and come and go during the progress of the fever.

In the third week the fever generally shows greater remissions, but the patient begins to show signs of exhaustion. With an average fall of perhaps a degree F. a day in the minimum temperature the upper range is often unchanged. The frequency of the pulse increases in severe cases to 120 to 140. The abdominal signs are more marked, and tremor, stupor, delirium, retention of urine, loss of sphincteric control, increased cough and albuminuria are common. It is at this time that hemorrhage from the bowels, perforation, hypostatic pneumonia, bed-sores and other severe complications are to be feared. In favorable cases the temperature subsides by rather sharp steps in the fourth week, with clearing tongue, disappearance of delirium, stupor, rash and diarrhea, slowing of the pulse and return of appetite. In severer cases the symptoms fail to clear up but continue into the fifth week, with running pulse, subsultus and muttering delirium. Relapse or some serious complication may occur just as the patient seems about to convalesce. No disease is more subject to unexpected changes.

The typical course outlined above is subject to so many variations as to render the diagnosis difficult in the extreme, although typical cases may be diagnosed practically at sight. A large proportion of the cases of enteric fever sent into the hospital are tagged "malaria," "influenza," and even in this day and age, "typhomalarial fever." This is in part because of the difficulty of diagnosis in the first few days but rather because sufficient care and skill are not used in the diagnosis. A further reason lies in the fact that the B.

typhosus may infect other than the usual tissues at the onset. The most common aberrant type is probably pneumotypoid, in which an almost typical pneumonia is present, but is later found to be due to the typhoid organism. In nephrotyphoid the symptoms at onset are those of acute nephritis. The cerebrospinal type of typhoid is commonly due to irritation of the meninges (meningotypoid) and in many cases passes for meningitis. Occasionally a true typhoidal meningitis occurs, with such localizing signs as to make the diagnosis reasonably sure without the recovery of the organism from the spinal fluid, or other proof. The occasional occurrence of afebrile typhoid or of only trivial elevation of temperature in the course of the disease should be noted. The occurrence of chills, especially in association with phlebitis, leads many practitioners into error, for the comparative frequency of this complication is not sufficiently appreciated. Typhoid septicemia, with severe course, delirium, perhaps petechiae and but little indication of the presence of intestinal lesions is extremely difficult of diagnosis in certain cases without the help of the laboratory.

**TEMPERATURE.**—That of the typical case has been sufficiently considered. The inverse type with the rise in the morning instead of the afternoon is not very common. Although the steplike rise is typical in those few cases seen from the very beginning, a sudden rise, perhaps following an initial chill is not at all rare and should not count against the diagnosis. Sudden falls, even of many degrees and not explicable by the occurrence of hemorrhage, phlebitis, pneumonia, or other demonstrable cause, are seen. A fall by crisis with immediate convalescence is not unknown. When the presence of typhus fever could possibly be considered, such a fall should excite suspicion.

The instability of the temperature after defervescence is notorious. Slight dietetic errors, anemia, starvation, visits of friends, constipation and persistence of an unhealed ulcer, etc., may cause a recrudescence of the fever. The instability of the heat regulating mechanism after a prolonged fever is well recognized. The exhaustion of the disease presumably accounts for the prolonged sub-normal temperature in many cases during an otherwise normal

convalescence. A slight persistent evening rise may disappear upon permitting the patient to get up and increase the diet. Temperatures exceeding 105° to 106° F. are unusual in typhoid fever and are significant of great danger. The temperature curve of the relapse is likely to be a mild imitation of that of the original attack, and subsequent relapses are often still milder.

*Chills.*—The initial chill is found in a few cases, though chilliness is not rare. The advent of a pronounced chill during the course of typhoid should suggest as the most frequent explanation the occurrence of phlebitis, often in the iliac veins, when the usual examination shows no trouble in the lower extremities proper. Next one should look for pneumonia, pleurisy, pyelitis or other complication. I have seen the plasmodium of malaria demonstrated as the cause of chills in typhoid in but a single instance. (Malaria is not indigenous in Colorado). When antipyretics of the coal-tar series were more frequently given in the treatment of typhoid daily irregular chills were not uncommon. A chill may occur after an injection of the prophylactic vaccine.

**CIRCULATORY PHENOMENA.**—In the early stages of typhoid, the dichrotic pulse is so common as to have a definite diagnostic value. In this region most cases presenting this feature turn out to be typhoid in the early stages or tuberculosis in rather advanced form. The pulse rate in general is decidedly increased though often not as much before the effects of exhaustion are superadded as is generally the case in fevers presenting an equal rise of temperature.

The gradual increase of frequency in the severe cases, with but little relation to the temperature curve, is to be noted. After the fastigium, when the temperature begins to fall the pulse may still be as high as 120 to 150 and in the severe cases may be uncountable. The blood count shows an absence of leukocytosis in the early stages, as is often seen in tuberculosis and in malaria, unless an increase is present from a septic complication. A relative decrease in the number of polymorphonuclear neutrophiles is noted, with some increase in the lymphocytes and transitionals. When pneumonia, perforation, etc., occur the usual polynucleosis is to be found. A

secondary anemia, of the usual type is common after the fever begins to subside.

Pericarditis and endocarditis are decidedly unusual complications in typhoid. On the other hand, changes in the myocardium are the rule, since the heart muscle cannot endure well the combination of starvation, overwork, and toxemia of a prolonged fever. Parenchymatous degeneration, often fatty, and perhaps accompanied by an interstitial myocarditis is common. Dilatation of the heart, relative mitral insufficiency, feebleness of the pulse, and of the heart sounds, pulmonary congestion, thrombosis, asthenia, and collapse all follow in the train of the myocardial changes. Thayer has shown the frequency of arteriosclerosis as a late sequel of typhoid, and degenerative muscular changes in the heart wall may become apparent years after the fever. Patients with valvular disease often do not withstand the strain of the fever, and acute dilatation and death may occur in the height of the disease. I have seen death in the second week from pure heart failure in a mild case of typhoid in a young woman who followed the then popular fad of "no breakfast and a diet of fruits." A well-nourished and efficient heart muscle is a *sine qua non* in the fight for life in typhoid fever.

The most common vascular complication is venous thrombosis, especially in the veins of the lower extremities, the femoral on the left side being the favorite seat. Although the right femoral vein may be affected alone, it is most often involved after the left has been obstructed. In some seasons thrombosis is far more frequent than the 2 per cent. to 4 per cent. commonly given as its ratio, would lead us to expect, and I believe it is undoubtedly more frequent under the tub bath treatment. I have seen six patients in successive beds afflicted at the same time. Comer believes that 10 per cent. to 15 per cent. of all cases of typhoid show venous thrombosis of some form, and that many of the pleural and pulmonary complications are of such origin. Thrombosis of the iliac vein often precedes that in the femoral and should carefully be sought for in event of pain in the lower abdomen, and especially if chills occur. Pain, chills, fever, swelling and tenderness are present. The organism of typhoid may be found in the clot. Pulmonary embolism may

occur from dislodgment of fragments of the thrombus. Pyemia is a rare occurrence. The swelling becomes less after a collateral circulation is established. If the iliac veins be involved, however, it is likely to remain a prominent feature for a long time, eventually subsiding somewhat as large venous communications develop above the groin on the affected side, carrying the blood around the obstruction. These great veins are so much more common as the result of typhoid than of other diseases that they offer semipresumptive evidence of its occurrence. In similar manner the presence of an enlarged left leg should lead us to ask first for a history of typhoid. Venous thrombosis is unusual in the arm, sinuses of the head, etc. The obstruction from thrombosis practically never causes gangrene.

Embolism and thrombosis of arteries, especially of the leg and arm, may occur in typhoid, as a result of endarteritis. Pain is more sudden and severe and gangrene shortly develops.

**RESPIRATORY PHENOMENA.**—A mild bronchitis is a part of the disease. Increased frequency of respiration attends the fever, apart from lung changes. Hypostatic congestion, and deglutition pneumonia are common complications of grave typhoid, coming on as the disease reaches its height.

Laryngeal ulceration is vastly more common in the autopsy room than it is thought to be by the average clinician. Although hoarseness, dyspnea, and even acute edema of the larynx may occur, a considerable lesion may attract no attention during the course of the disease. A laryngeal neuritis is a rare sequel to typhoid.

Lobar pneumonia may occur, as mentioned above, as a manifestation of the localization of the typhoid bacillus in the lungs, a true pneumotyphoid, indistinguishable at first from acute pneumonia. The failure of crisis and the development of abdominal signs and symptoms of the disease lead to a correct diagnosis in the second week. Laboratory tests greatly hasten the diagnosis in this form.

During the height of the fever a complicating lobar pneumonia, due to the usual organisms of the disease, may develop with chill, pain, and fever, expectoration and other signs and symptoms, all rendered less distinct by the characteristics of the primary disease. Sudden increase in the respiratory rate should always suggest it.



The usual complications of lobar pneumonia may follow. Pleurisy, apart from that form complicating the lobar pneumonia, may develop, either as a dry form, or with serous or purulent effusion.

Hemoptysis is rare. It should suggest the possibility of a concomitant tuberculosis. An attack of typhoid fever of any severity is a grave thing in phthisis, often starting the original disease upon a more active course. In this region, to which so many tuberculous individuals are attracted, especial care should be used to keep convalescing typhoid patients from exposure, owing to lack of resistance which they offer to infection. A wholly latent tuberculosis may become active even apart from a new exposure.

CUTANEOUS MANIFESTATIONS.—The rose rash is found in 75 per cent. or 80 per cent. of well-observed cases. A half dozen or more rosy, slightly elevated, palpable spots, 2 or 3 mm. in diameter, may be found upon the abdomen, and lower chest in most cases, and with nearly as great frequency over the loins. In probably 1 per cent. or 2 per cent. of cases the rash extends to the limbs, notably to the upper portions, and rarely to the face. Yet I have twice seen it suggest the facies of typhus fever from its abundance.

The eruption generally appears on the sixth or seventh day, and the individual spots come and go during the disease. They should be encircled with a pencil mark, for future reference, if the diagnosis be still in doubt. Their chief diagnostic feature is their disappearance upon pressure. The Eberth bacillus may be isolated from blood drawn from the spots. A new crop often appears with a relapse. The rash is of great value in diagnosis if taken in conjunction with other features, but a few reddish spots apart from these features are of no value, since they may be due to many causes.

In cases which perspire freely sudamina are common, but of little significance. The tache cérébrale is of little value in diagnosis. The absence of herpes labialis is notable and of some value in the differential diagnosis, between typhoid on the one hand and pneumonia, cerebrospinal meningitis, or malaria on the other hand. Urticaria, erythema, peliomata (from body lice), and desquamation may be noted. The skin of the palm is often yellow. The odor of the dis-

ease, dependent in part upon the skin secretions, is almost sufficient for the diagnosis in some cases.

More important than any other skin manifestation, excepting the rose spots, is the occurrence of a purpuric eruption. I have seen it in two forms,—as diffuse, large petechiæ in the severest and most septic type of the disease, and, so far as I know, always a fatal indication; and as small, purpuric spots, sometimes in connection with the ordinary roseola, and of much less grave omen. Four of the seven cases of which I have notes, seen in a total of something over 1,000 cases, recovered. Furunculosis is not rare after typhoid. Local gangrene, apart from that of bed-sores, is occasionally noted.

**NERVOUS SYMPTOMS.**—In the first days of the fever the headache may be the most prominent symptom, gradually becoming less so until, with the advent of the dull stuporous state in the second week, it disappears. Stupor and delirium then become features of the disease. The delirium is at first chiefly nocturnal and rarely violent. The moderate deafness of typhoid associated with a delirium from which the patient may be roused for a moment is fairly characteristic of the disease. The delirium in which the patient has some fixed idea, as of going home, is ominous. In drinkers, delirium tremens may appear as a complication. Although typhoid delirium rarely assumes an active form, the patient must be carefully watched, since attempts to escape are not uncommon. The deepening of the stupor and the delirium to the degree that the patient cannot be aroused is of gravest significance, and when coma vigil, subsultus, and carphologia appear a fatal result may be looked for. Marked tremor of hand and tongue may precede these symptoms for several days.

In so-called meningeal typhoid, the patient may present all the signs of meningitis, excepting the localizing ones affecting the cerebral nerves, and yet have only a congestive irritation, as shown by the complete recovery. Many cases diagnosed as meningitis in children are undoubtedly typhoid, as shown by the occurrence of other cases diagnosed as typhoid in the same family or epidemic. I have known a patient to hold her head in her hands and scream for a week, so that she had to be isolated, without any sign of true meningitis, and with full recovery. Nevertheless a true meningitis, due

to the typhoid bacillus, or to other organisms, if sepsis has occurred as a complication, is occasionally seen. Lumbar puncture should be performed if doubt exists.

Convulsions occur in children at the onset occasionally, and rarely in adults, as a fatal termination. It is likely that the toxemia of the disease causes the latter form in some instances, although thrombosis of the cerebral vessels is more probable. Hemiplegia may occur as the result of cerebral vascular disease, or meningo-encephalitis.

Multiple neuritis of the ordinary post-febrile type is occasionally seen involving the arms and legs, or but a single member. Pain, tenderness, disability, and perhaps swelling may be seen. The extensors are more involved than the flexors, so that the wrist-drop and foot-drop may be noted. The affection spoken of as "tender toes" doubtless depends upon a mild local neuritis (often in association with thrombosis of veins in the sole of the foot) and disappears in a week or two. Post-febrile exhaustive insanity occurs more frequently after typhoid than after any other disease, but only in a small fraction of one per cent. of all cases. Melancholia is the most frequent manifestation of the trouble, as I have seen it, the patient often refusing food and suffering from various hallucinations. It occurs particularly in patients who have recovered from a long exhausting attack, with pronounced nervous manifestations. Fortunately, with proper feeding and care, the prognosis is quite good, as in other types of exhaustive psychoses.

**BONY COMPLICATIONS. TYPHOID SPINE.**—This complication, formerly thought to be always a neurosis, is shown by the use of the X-ray to be often accompanied by periosteal changes, fortunately not going on to suppuration as a similar process in the ribs, tibia, etc., often does. Goldthwait believes that many of the cases showing no changes with the X-ray are attributable to the strain of the spinal ligaments, from improper support of the back during the long illness. Pain in the back, especially upon movement, often with rigidity and deformity, is the prominent feature. Pain often extends to the legs, owing to pressure upon nerve roots. The prognosis has much improved in my own cases since the use of mechanical supports for the back as in cases of osteoarthritis.

Arthritis, due to the specific organism, or to complicating infection, is occasionally seen, and commonly affects but a single joint. A periostitis of similar origin is more common, the "fever sore" of the laity, affecting especially the ribs, costal cartilages and tibia. Superficial necrosis of the sacrum or other bone exposed by a bed-sore occasionally occurs, and delays convalescence, for the bony lesions are notably slow to heal.

**SYMPTOMS PERTAINING TO THE ABDOMINAL ORGANS.**—Diarrhea is the most frequent of these, being present in rather less than half of the cases at some stage. It is largely dependent upon the congestive and ulcerative conditions within the ileum. The stools, not generally over 5 to 8 in the 24 hours, are thin, yellowish, foul, alkaline, and commonly contain the specific organism of the disease after the first ten days or so. Masses of milk curd, small sloughs from the intestinal mucous membrane, blood in microscopic quantities, or in greater abundance, are often seen.

Vomiting is sufficiently rare in typhoid to raise a presumption against the diagnosis, yet it occasionally occurs. Hematemesis is rarely seen.

*Tenderness and Pain.*—The former is the more frequent of the two, and is especially noted in the right iliac fossa, but often only when especially sought for. If low down, particularly upon the left side, iliac thrombosis should be suspected. Pain is also often present in this event. The complaint of pain should always suggest an over-distended bladder, so common in this disease, and, if sudden, the possibility of intestinal perforation. Pleurisy, pneumonia, and cholecystitis are other common causes of pain and tenderness in the abdomen. The spleen, while enlarged in most cases, is rarely tender.

*Hemorrhage.*—This occurs in 3 per cent. or more of all cases, varying much in frequency in different epidemics. About 40 per cent. of those cases affected are fatal. The bleeding of the ulcer at the time of the separation of the slough is commonly noted in the third or fourth week, and varies from a mere streak to hemorrhage of such severity as to cause death before the blood has time to escape from the bowel. The streak of blood upon a formed stool is presumably from a mere oozing about the inflamed Peyer's patch, and

may be seen in the second week. I have known several hemorrhages to occur in a patient on the street a month after he was up and about, possibly from a single unhealed ulcer, which failed to give any other evidence of its presence. In the cases of hemorrhagic type the intestinal hemorrhage may be of the same purpuric origin as the petechiæ and the bleeding from the gums.

A considerable hemorrhage is generally accompanied by a sharp fall in temperature, this being often noted before the blood has time to escape from the anus. Symptoms of faintness and collapse are often present. If the blood be long retained it is tarry in appearance when passed. The possibility of perforation coincident with the hemorrhage should be borne in mind.

*Intestinal Perforation.*—Intestinal perforation is the cause of death in 3 per cent. to 5 per cent. of all cases, unless prompt surgical intervention lessens this rate. It has been less amenable to amelioration by modern medical treatment of typhoid than the other causes of death. It is to be looked for after the middle of the second week when the sloughing process is at its height. Mildness of the attack offers no immunity. It is more common in cases with diarrhea, abdominal distention, and hemorrhage.

The first symptom is ordinarily the sudden pain, like that of any other perforative intestinal or gastric lesion, but commonly somewhat covered up by the dulled mental condition of the patient. The advent of pain may be gradual, if the perforative process be in part walled off. Since the part of the ileum most seriously involved in typhoid lies chiefly in the right iliac fossa, the pain is likely to be felt there. Next there is local tenderness, followed by rigidity, both speedily becoming general. The intense boardlike rigidity, differing entirely from the distention of tympanites, is perhaps the most certain sign of the perforation, really indicating the peritonitis which follows it. Collapse, sweating, increased frequency of the pulse, increase in blood tension, and frequent urination may follow. In patients with marked stupor the abdominal rigidity may be the first sign of the complication. A gradual fall of the blood pressure from the normal of about 125 mm. to 110 to 100 mm., may be noted in most cases of typhoid, and an increase from what has become the

temporary normal during the typhoid is very suggestive of the rise which occurs with perforation. Frequent examinations should be made in suspicious cases.

As the peritoneal inflammation progresses, we have distention of the abdomen (occasionally absent), escape of gas from the bowel into the free cavity, with obliteration of the liver dulness and occasionally that of the spleen, accumulation of the exudate in dependent parts of the abdomen, friction, audible and palpable, as the roughened peritoneal surfaces move over each other, absence of peristaltic movements, often increase in temperature, the development of polynucleosis, and, finally, collapse, facies hippocratica, and death. Occasionally a typhoid perforation is followed by a walled-off abscess and recovery. In one of my cases such an abscess was opened in the ischiorectal region, with recovery.

The symptoms and signs of perforation may be closely imitated by the peritonitis which develops in advance of an extending ulcer which yet does not actually perforate. Local peritonitis may occur by infection through the base of an ulcer without gross perforation, as in gastric and duodenal ulcers. Typhoid ulcers occur so frequently in the appendix that the symptoms of appendicitis may occur in the midst of typhoid, there being in fact a *typhoidal appendicitis*. In several cases of this nature which I have had operated, the symptoms of appendicitis overshadowed those of the typhoid, and in cases of suspected perforation, we have sometimes found large, tense, shiny lymph glands, which I do not doubt, in the absence of perforation, have been responsible for the symptoms. We cannot properly wait for absolute certainty of diagnosis in these cases, since the recovery rate is proportionate to the promptness of surgical intervention.

*Liver.*—Hepatic abscess is recorded in typhoid, but is rare. Jaundice occasionally develops. Cholecystitis is frequent as a complication of typhoid, and probably in certain cases of the disease the infection spends its force in the gall-passages. The enlarged gall-bladder may be felt in certain cases, and rigidity and tenderness are marked. I have seen subphrenic abscess with subsequent left-sided empyema (operation with fatal result) as a result of perforation of a neglected cholecystitis. The infection in the pleural cavity was due

chiefly to the colon bacillus in this case. Perforation may occur into the free peritoneal cavity with symptoms like those of intestinal perforation. Suppurative cholangitis is not common. Most cases of cholecystitis recover, but the frequency of the history of typhoid in gall-stone disease testifies to the part played by the *B. of Eberth* in bringing about cholelithiasis. The organisms have been isolated several decades after the fever. The spread of typhoid by "carriers" is often due to the fact that the bacilli persist in the bile passages after recovery from typhoid (2 per cent.), and the stools thus remain infectious indefinitely. Gall-stones may be present meanwhile.

The spleen may rupture from excessive distention, or from abscess formation after infarction.

The tongue is coated in the early stages of the fever, but as the stupor increases becomes dried and cracked from the mouth-breathing, being often brown or even black. The sordes collected upon the teeth represent the dried blood exuded, sputum, epithelial cells, and various micro-organisms.

**EYE, EAR, ETC.**—Oculomotor paralysis has been observed. In general, apart from conjunctivitis, the eye escapes in typhoid, though iritis and corneal ulceration may occur. It is common for patients to first suffer from inability to use the eyes without glasses after recovering from typhoid, being unable, because of lack of strength, to overcome defects in vision which were formerly scarcely noticeable. I have seen persistent headache from eye-strain in convalescence from typhoid fever, caused by the glaring light reflected from newly plastered walls, with immediate relief upon introduction of efficient window shades.

Otitis media is fairly common and mastoid suppuration may result. Because of the patient's mental dulness the ear complications must be watched for carefully.

Parotitis is fairly common, and I believe less fatal if operated upon early than is commonly thought. I have seen recovery after perforation through the canal of the ear, and repeatedly after surgical opening, even when both glands were involved. The inflammation probably occurs through the duct from the mouth, offering an additional argument for keeping the mouth as clean as possible.

Bed-sores are most common in severe, neglected cases, but may

occur despite every attention. The sacral form is hardest to avert, while those of the heel are easiest, being commonly avoidable by supporting the calf upon a pillow with the heel free. I have seen decubitus also upon trochanters, over the scapulæ, occiput, internal condyle of the elbow, head of the fibula, internal condyle of the knee, inner and outer surface of the ankle, and over the dorsal spines. Sepsis and death are not uncommon if the bone be extensively uncovered. Gangrene of patches of the skin of the back or the genitals, of a dependent external pile, and of the cheek (noma), with death from perforation of the facial artery, have fallen under my observation. Painful callous formations, "corns," upon the ear are occasionally annoying in convalescence. Stone masons and others with calloused hands often complain much upon recovery of the loss of the protecting thickened skin, which has occurred during the disease and which takes weeks to reform. The "lineæ albicantes" upon the abdomen, and occasionally over the hips and shoulders of young individuals, especially females, are found after recovery from typhoid. I have never seen them, excepting when the skin has been stretched with the accumulating fat of convalescence.

Rupture of the recti muscles with hemorrhage, and even abscess formation, may be seen as a result of the degenerative process accompanying typhoid.

Acute nephritis has been mentioned. Hemorrhage from the kidneys may occur in the purpuric cases. Albuminuria with tube casts is to be expected in the severe cases. Pyelitis with passage of the bacilli in the urine is not unusual, and represents a common cause of the dissemination of the disease. I have seen several of these cases operated years after recovery, the pyuria having been often more or less intermittent in character, with repeated accumulation of purulent fluid followed by emptying of the pelvis. Cystitis is not uncommon, and, like pyelitis, is dependent to some extent upon neglected retention of urine. The bacillus coli is perhaps more common than the bacillus of typhoid. Repeated use of the catheter naturally favors infection. Orchitis occasionally develops, even apart from a urethritis.

Women often fail to menstruate during the disease, but occasionally flow too freely. Mastitis may occur.



**RELAPSE.**—This occurs in a considerable number of cases, variously stated as from 5 per cent. to 15 per cent. by most authors. A second relapse is not rare. I have seen three, and even four and five have been noted. It is more common in the use of the tub bath treatment, in part at least, because more patients survive and become liable to relapse.

A relapse proper begins some days, or even two or three weeks after the temperature has become normal, and has usually a milder and shorter course than the original disease. It presents the fever, rose spots, enlarged spleen, and other features of typhoid. The recrudescence of fever for a day or two does not constitute a relapse. An intercurrent relapse, as the name indicates, is one starting in before the original fever has subsided. It is probable that the cause of relapse is a reinfection from within, presumably from the bile passages, and that the occurrence of relapse after solid food is given is merely because the increased digestive action called for leads to the discharge of infected bile into the intestines. It is necessary to assume that the immunity which typhoid commonly confers later has not yet been established.

**Diagnosis.**—During the first week of typhoid the diagnosis is usually impossible, excepting in case of an epidemic or recognized exposure, unless by means of the blood culture. In case of pneumo-typhoid, nephrotypoid, typhoidal sepsis, and the meningeal form early diagnosis is rarely possible. Typhoid in some of its forms resembles many other diseases, and the less common diseases are ordinarily thought to be typhoid in the beginning. Tuberculous meningitis, septic endocarditis, and pyemia are examples. If there be a history of a well-defined attack of typhoid the presumption is against its recurrence.

A careful investigation of possible opportunities for infection is of some value in the diagnosis and of great value in prophylaxis. I have seen this week a girl who drank at a certain well at a picnic, and three companions who also drank from it, come down with typhoid within two weeks thereafter. Such a history strengthens the provisional diagnosis in her case.

The important symptoms upon which we may base a diagnosis at

the bedside are: the characteristic temperature, the enlarged spleen, and the eruption. Yet it is a week before these *are characteristic*. The presence of a dichrotic pulse, of a pulse rather low in proportion to the temperature, of the diazo-reaction, nosebleed, coated tongue, and diarrhea add to the probability of typhoid. The occurrence of intestinal hemorrhage is often the first decided evidence in cases which the physician has had little opportunity to observe. In case of doubt in a continued fever, it is best for the physician to place himself upon the side of the probable typhoid rather than to remain long upon the defensive, for the treatment necessary in typhoid is probably the best that can be offered until the diagnosis is definitely established.

At the end of the first week the Widal reaction will probably be positive (more than 90 per cent.), and the diagnosis is then settled, provided the patient has not recently had an attack of the disease. The procedures for the finding of the bacilli in the blood, urine, and in the stools by the Drigalsky-Conradi method should be taken advantage of when possible if doubt exists. The ophthalmic reaction has not been generally adopted. The bacillus is not present in the urine until the third week in most cases.

**Differential Diagnosis.**—At the time when typhoid fever shows little except malaise and a gradually rising fever, it may easily be confused with some of the eruptive fevers. The sore throat and early vomiting and rash of scarlet fever, the coryza, bronchitis, and eruption of measles, and the chill, vomiting, backache, and shotty eruption of small-pox prevent any long continued doubt as to these diseases.

During the early days, febricula must be considered. It is so transient an affection that its failure to steadily increase in severity for two or three days should lead to its exclusion. In febrile influenza there are generally more catarrhal symptoms, often conjunctivitis, less of the dull expression of typhoid, commonly absence of the abdominal symptoms, and defervescence at the end of six or eight days.

The "gastric fever" of certain practitioners, so far as I have observed, runs a course like that of a mild typhoid, and the finding of

the Widal reaction in many such cases has led to the abandonment of the term.

**APPENDICITIS.**—If the pain of the attack be not too severe, and the febrile manifestations be marked, the disease is often mistaken for typhoid. When we recall the comparative frequency with which a typhoid ulcer is found in the appendix, we need not be surprised at the frequency of the false diagnosis of appendicitis as an independent disease, since we have in such a case a typhoidal appendicitis. Ten per cent. of the typhoidal perforations in McCrae's series were in the appendix. Greater abruptness of onset, greater pain, rigidity and tenderness, frequent vomiting, less degree of fever, and more rapid development characterize appendicitis. The diagnosis is likely to be settled before laboratory methods of diagnosis become available, unless a sharp leukocytosis develops.

Much criticism is heard as to operation in early typhoid for wrongly diagnosed appendicitis, but with the greatest care it is impossible to avoid an occasional error. Thus in one of my cases of undoubted typhoid the typical symptoms of appendicitis were superadded, and perforation was thought to be present or imminent. At operation a typhoidal ulcer in the appendix had so nearly perforated that lymph flakes covered the surrounding loops of bowel, and a small amount of cloudy fluid was present. The patient's course toward recovery was uninterrupted. In another case a girl of seventeen had had typhoid two years before with a typical Widal reaction. Her sister in the next room had typhoid. She was taken suddenly ill with fever, pain, tenderness, rigidity, and vomiting, and nothing would have suggested typhoid, excepting the presence of the disease in the house. Against this was the known history of typhoid two years before. After anxious consideration, with my advice, she was operated upon only 48 hours from the acute onset. A typical typhoidal enlarged mesenteric gland came into view near the incision and a typhoidal ulcer was present at the middle of the appendix. Both of these cases ran all the risks of appendiceal perforation, in addition to those of enteric fever, and both were saved by the application of the rule that if the appendiceal symptoms are clearly marked, and would demand operation apart from the presence of typhoid,

operation should be performed. Unless, however, it is very clear that a sharp localization at the appendix is present, it is safer to assume that the symptoms are due to typhoid lesions, and to await further developments. I could quote further experiences where the finding at operation confirmed the position above stated. In the type of appendicitis in which the organ lies upward, behind the cecum, physical signs of its disease may be very doubtful, and yet it is in this very type that ascending infection leading to liver abscess, sub-phrenic abscess, etc. so often occurs. I have known several such cases, treated as typhoid, until the occurrence of edema, bulging of the liver region, etc., attracted the attention. After seeing nearly a score of cases of this type of appendiceal affection operated, I erred for three or four days in one case, concluding that the case was typhoid. Operation then demonstrated the error.

PNEUMONIA.—It is impossible to diagnose the rare pneumotyphoid at the beginning. I showed one at my clinic with every typical feature of acute lobar pneumonia, aside from bacteriological evidence, and not until the rash came out did I suspect the error, if it can be called an error to show, as pneumonia, a case in which true pneumonia exists, but which has a bacteriology differing from the ordinary form. In the aged the physician may pay so much attention to the pneumonia which develops so often as a complication, that he overlooks the original typhoid fever. Ordinary acute pneumonia can scarcely be confused with typhoid after the first day or two.

TUBERCULOUS MENINGITIS.—This disease is commonly suspected to be typhoid within the first few days, when no decisive signs are present. The preliminary poor health, less degree of fever, scaphoid abdomen, frequent vomiting, slower course in many instances, and the severity of the headache, generally lead us to a correct diagnosis, before the advent of Kernig's sign, rigidity of the neck, ocular paralysis, or evidence from the examination of the spinal fluid. The generalized miliary form of tuberculosis offers more trouble in exclusion, since the meningeal signs and symptoms may not be present to assist us. The increase of the respiratory rate even to 60 or 80 per minute, with cyanosis, and less physical evidence in the chest than one would expect, should excite suspicion. The diazo-reaction may be

present in either disease. The Widal test should be tried and repeated if there be a negative result, for the positive reaction is decisive. On the other hand, the positive result of a tuberculin reaction is of no value, since many children with typhoid may still react, owing to the presence of tuberculous bronchial glands or other foci of infection. Tubercles in the choroid occasionally offer decisive evidence. Spots upon the abdomen, indistinguishable from the eruption of enteric fever, add to the confusion in the early days. Departures from the rule as to the type of fever and the general character of the pulse, suggest the tuberculous disease. As is so often the case in diagnosis, time is necessary before certainty can be obtained.

In tuberculous peritonitis the abdominal distention, diarrhea, irregular fever, and rapid loss of flesh lead to the suspicion of typhoid, but the greater mildness and chronicity of the disease, the doughy feeling of the abdominal wall, the presence of effusion in the abdomen, practically never seen in typhoid, excepting after perforation, are sufficient for its exclusion. Foci of tuberculosis elsewhere, and especially the presence of palpable glands, aid us in certain cases.

**CEREBROSPINAL MENINGITIS.**—From the clinical features it is impossible in many cases to differentiate between this disease and the meningeal type of typhoid, since the symptoms of each are dependent upon congestion, irritation, and inflammation of the meninges. The two diseases may have in common sudden onset, vomiting, headache, photophobia, purpuric eruption, retraction of the muscles of the neck, Kernig's sign and delirium. The examination of the cerebrospinal fluid for the meningococcus, the presence of a polynucleosis, the absence of Widal reaction and the development of herpes labialis, with great rapidity in the evolution of the disease, point toward the cerebrospinal fever. Rose spots and abdominal symptoms lead rather to the diagnosis of typhoid.

**ULCERATIVE ENDOCARDITIS.**—Perhaps a majority of the cases of this disease are at first taken for enteric fever. In ordinary cases there are sufficient physical signs to be found upon careful auscultation and percussion of the heart to prevent error. Presence of leucocytosis, absence of Widal reaction, possible presence of minute reddish spots, indicating the lodgment of small septic emboli, and the

absence of any data essential to typhoid aside from the fever and the enlarged spleen, should prevent error. I have seen the heart dilated to a point beyond the nipple line with a loud mitral systolic murmur, high fever, abundant petechial eruptions and large spleen, and death before the blood was examined. My diagnosis was malignant endocarditis, but autopsy proved it to be a case of most typical fulminating hemorrhagic typhoid fever.

**SEPTICEMIA AND PYEMIA.**—If there be an obvious cause for such trouble no difficulty should be experienced. In the cryptogenetic form, as in obscure appendiceal, mastoid, prostatic or hepatic abscess, confusion occurs. The various blood tests are of the most decisive value, and should suffice for a correct diagnosis. Various septic complications of typhoid may, if seen for the first time, obscure the original disease, unless a careful anamnesis be secured. The septic type of typhoid has been mentioned elsewhere.

**MALARIA.**—The frank forms with typical paroxysms are easily excluded. In the type of *æstivo-autumnal* fever, seen more often in the southern half of the United States, in which a more or less continued fever is present, without chills, and often with diarrhea and enlarged spleen, the demonstration of the parasite in the blood may be and often is necessary for the diagnosis. The presence of the Widal reaction, and the lack of effect from quinin, suggest enteric fever, while the presence of herpes labialis suggests malaria. In case of doubt, if quinin can be safely withheld, the chances of demonstrating crescents or other decisive evidence of malarial infection are greatly improved. The possibility of the presence of both typhoid and particularly the *æstivo-autumnal* variety of malaria in combination must be borne in mind. Although not rare in warm regions I have known but a single instance in Colorado. The term “typho-malarial fever” inherited from the surgeons of the Civil War, is obsolete.

**TYPHUS FEVER.**—Excepting at quarantine stations, this disease has been practically unrecognized in this country until the recent work of Brill in New York. In the face of an epidemic the diagnosis is comparatively easy, attention being attracted by the more sudden onset, more early and severe delirium, greater stupor, congested con-

junctivæ, and the petechial eruption on the fourth day. The termination of the fever by crisis in about two weeks is very striking. The eruption upon the face in typhoid may be as abundant as that which may occur in typhus, but lacks the petechial character, unless in rarest instances. The most impressive features in diagnosis in the small epidemic of typhus which I saw were the injected conjunctivæ, abundant eruption, with petechiæ, and swollen appearance of the face. The blood examination should be resorted to in case of doubt. Correct and early diagnosis is of supreme importance because of the contagiousness of typhus.

Relapsing fever, anthrax, uremia, and trichiniasis are to be thought of but may be excluded as a rule without difficulty. Salpingitis and other pelvic troubles should be excluded if a pelvic examination be carried out. In nephrotyphoid the nephritic features tend to clear up and give place to those of the general infection.

**DIAGNOSIS OF PERFORATION.**—Recovery from perforation in typhoid depends almost wholly upon early diagnosis and efficient surgical measures. In all cases, therefore, and especially in those with tympanites, diarrhea and hemorrhage, nurses and house physician should be directed to watch carefully for the earliest signs of the accident. Sudden pain, especially in the lower right quadrant of the abdomen, gradual increase of rigidity, tenderness, shock, hic-cough, nausea and vomiting, and the gradual increase of the blood pressure, as shown by the sphygmomanometer, with wiry pulse, are the important data. A rectal examination occasionally shows exquisite tenderness. After collapse and disappearance of liver and splenic dullness and presence of effusion in the peritoneal cavity are to be found, operation will be of doubtful value.

On the one hand, perforation may occur from any of the other usual causes during the course of enteric fever, while on the other, rupture of the spleen, enlargement or softening of the mesenteric lymphatic glands, development of local peritonitis, in advance of the deep ulcerative process in a Peyer's patch, intussusception, or other intestinal accident, or iliac phlebitis, may deceive us. Absolute diagnosis is impossible in most cases without loss of time, and if the signs and symptoms show that the trouble is getting worse, early operation

is indicated. With a working diagnosis and early operation we may save the patient; with an absolute one and late intervention, death is to be expected. The diagnosis should not be clouded by the giving of opiates.

**Prognosis.**—The death rate of typhoid before the introduction of the tub treatment was commonly ten to eighteen per cent., as shown by statistics from the entire civilized world. It may be reduced by tubbing to about seven to eight per cent. under favorable circumstances, as they affect the “character of the risk,” opportunity for early and efficient treatment, careful watching for perforation, with prompt surgical aid, etc. In general the prognosis is worse with added years, with obesity, pregnancy, alcoholism, and any type of chronic disease. Rapid pulse, severe nervous symptoms, and hemorrhage are serious signs, and perforation without early operation is almost always a fatal event. The earlier any serious symptoms develop, the worse the prognosis.

The mortality amongst the 6,948 cases reported in the years 1902, 1903, and 1904 to the Colorado State Board of Health was fifteen per cent. In a State so sparsely settled and with few cities, many mild cases escape report, so that this rate is unduly high. I speak with greater confidence upon this point because of twelve years of official connection with this board.

### PARATYPHOID FEVERS

These were first recognized only by the failure of the infecting organisms to react as the *B. of Eberth* is known to do. The organisms are classified between the typhoid bacilli, on the one hand, and the colon bacilli on the other. We may quote Buxton's classification as follows:

**“Paracolons.**—Those which do not cause typhoid symptoms in man. A group containing many different members, culturally alike.

**“Paratyphoids.**—Those which cause typhoidal symptoms. (A) A distinct species culturally unlike the paracolons. (B) A distinct species culturally resembling the paracolons. The relative frequency of forms A and B is stated to be as one to five. Paratyphoid A



resembles typhoid more closely, while paratyphoid B may resemble the septicemias, the infection from *B. enteritidis*, or acute meat poisoning."

Typhoid varies much more in its different varieties than do the paratyphoid cases from typical enteric fever. No pretention to differential diagnosis is made without the application of the agglutination test. The ordinary type of typhoid, and the septicemic types are most frequently imitated. The organisms in question may be found in abscesses apart from known typhoid fever. The ordinary type of paratyphoid fever is likely to be mild, with recovery. In the septicemic form, enlargement of the spleen and absence of the pathological characteristics of typhoid are the chief findings at autopsy.

The diagnosis of the paratyphoid fevers is then a laboratory procedure and leads to no modification of treatment unless it proceeds from the laboratory.

## II. TUBERCULOSIS

**Definition.**—This is an infectious disease caused by the bacillus tuberculosis, and found either in the acute, subacute or chronic form. Formation of tubercles, infiltration, caseation, necrosis, and ulceration are successive steps in the pathology of the disease, although it may be arrested by processes of sclerosis and calcification.

**Etiology.**—Tuberculosis is the most wide-spread of human diseases, and affects many of the domestic animals as well. About one-seventh of human deaths are due to it. Kaiserling stated at the Paris Tuberculosis Congress that in Germany one-third of all deaths and one-half of all sickness amongst adults are chargeable to this infection. In seventy to eighty per cent. of the cases the lungs are the seat of the disease. Tuberculosis is especially favored in its development by causes which undermine the resisting power, such as over-crowding, under-feeding, lack of sunlight and fresh air, inheritance of poor tissue resistance, dissipation, mental depression, etc. Amongst other factors favoring its development, apart from special exposure from association with those infected, which is of course a predominating feature in the etiology, may be mentioned:

(1) Sex; females being rather more susceptible; (2) age, early adult age being the time of most frequent infection, especially in the pulmonary form; (3) race, Indians and negroes being especially susceptible, perhaps from lack of long racial exposure and consequent partial immunity. In America, foreigners and mulattoes have a higher tuberculous death rate than the native Americans, but doubtless largely from greater exposure in occupation, and less resistance because of unfavorable surroundings. (4) Occupation. Those exposed to dust and confined air are often infected, as in cotton factory operatives, janitors, millers, etc.; in many cases, notably in miners, a chronic bronchitis may exist for years from the irritative effects of stone dust, particles of steel, powder smoke, gases from other explosives, etc., and the tuberculosis may be a late secondary involvement. (5) Previous diseases, especially those involving catarrh of the respiratory tract, such as measles, whooping-cough, typhoid fever, small-pox and especially influenza. (6) The debility resulting from chronic wasting diseases (diabetes, hepatic cirrhosis, syphilis, chronic nervous disease), in many of which the tuberculous infection is a terminal process. Lessened resistance and increased opportunity for infection both play a part here.

Amongst those conditions favorable for avoidance of infection with tuberculosis may be mentioned good heredity and good physique, a good digestion, and good food; residence and occupation involving out-of-door life, and consequent avoidance of dust and bad air; dryness of soil and of climate, abundance of sunlight, elevation above the sea, sparse population, with its consequent lack of opportunity for infection and for contamination of the air with products of respiration and combustion. In those regions where the conditions are all generally favorable, as in the Rocky Mountain Plateau, where sparseness of population, dry soil and dry air, elevation, favorable out-of-door occupations and easy attainment of good food all contribute to safety, tuberculosis is practically unknown, excepting as it is imported, or as it occurs in those especially exposed.

Contrary to the popular opinion, especially of a few years ago, it is found that residence in or near resorts frequented by tuberculous patients not only does not predispose to the disease, but, because

of better knowledge of methods of prevention and better hygienic surroundings, that tuberculosis is actually less frequent in these localities. In general we may state that heredity is looked upon as playing a vastly less important rôle than was formerly thought to be the case, and opportunity for infection in a family having active cases of the disease is believed to be much more important. The Jewish race is much less susceptible to tuberculosis than other peoples.

In animals the frequency of tuberculosis in those in confinement, as contrasted with its rarity, or probable absence, in the wild state, is very striking. Tuberculosis is the chief cause of death of the inmates of zoölogical gardens and menageries. Milch cattle kept in confined quarters, and especially the varieties giving rich milk (Jerseys) suffer terribly from the disease. The cattle raised on the Western plateau, on the other hand, are almost exempt from it.

**The Causative Organism.**—The tubercle bacillus is to be found in all the lesions of tuberculosis with greater or less ease according to the tissue, and the activity of the process. Bacteriologists recognize four varieties of organisms, dependent, largely, at least, upon the host—the human, bovine, avian, and piscine and reptilian types. The tendency of recent years is toward the belief that these varying types are essentially the same, and that the variations depend wholly upon the animal host. There seems to be no room for doubt, in view of recent reports based upon very extensive investigation, that infection of man by the bovine type of bacillus is not only possible but frequent enough to be of great importance. Thus the tuberculous glands of the neck are stated to be usually of the bovine type. For the consideration of the varieties of the tubercle bacillus and of the score or more of the so-called pseudotubercle bacilli, and of the staining, and the cultural biology and biochemical investigations of the organisms, the reader is referred to the special works upon tuberculosis.

**Infection.**—Man is rarely infected by direct hereditary transmission; occasionally by inoculation, as in the post mortem room, and in the handling of the sputum cups, etc.; more frequently by ingestion of infected food, notably of milk and its products, and of meat; and even more often, probably, so far as our present knowledge goes,

by inhalation of the infecting organism, as by direct exposure to the droplets coughed out by patients with pulmonary tuberculosis, or especially by exposure to dust which has been rendered infectious by the lodgment of masses of sputum, droplets of sputum, other secretions infected with the bacillus, etc., and which, upon inspiration, carry the bacilli directly into the respiratory tract. The great frequency of endemics of tuberculosis in shops, factories, offices, etc., where promiscuous expectoration has been permitted and where tuberculosis has been recognized in some of the inmates, need only be mentioned. The frequency of the disease in certain schools, prisons, in quarters of certain religious communities, and even in private houses, in the families of successive occupants, and the almost constant tuberculous involvement of the tracheobronchial glands of children exposed in foundling asylums, etc., as shown by post mortem investigations (Northrup), are features pointing to the ease and frequency of infection through the respiratory tract. In similar manner the frequency of infection of the bowel and mesenteric glands in children living largely on milk, points to the danger of infection from this cause, and numerous feeding experiments have shown the ease with which infection may be produced in animals. In babies crawling upon the floor the danger of infection from the carrying of dust to the alimentary canal by means of the soiled hands is probably a grave one.

#### A. ACUTE MILIARY TUBERCULOSIS

For convenience in description it is customary to arrange the different types of miliary tuberculosis under the separate headings as follows:

- (1) General or typhoidal form;
- (2) Pulmonary form;
- (3) Meningeal form.

The methods of infection and the general features of the different forms may be first noted, since these apply in some degree to all.

An infection often occurs suddenly in many different organs, analogous to the general infection in septicemia. The different organs

and the serous membranes are thickly studded with miliary tubercles. The symptoms are modified in accordance with the varying predominance of the infection in different regions, and the acuteness of the infection.

The source of the infection is practically always some degenerating tuberculous focus, and the bacilli are spread broadcast by the circulation, access to a vein, artery, or even the thoracic duct, having been afforded either by rupture of the focus through the vascular wall into the stream, or through the eruption and degeneration of a tubercle in the intima of the vessel. Many cases originate from broken-down tracheobronchial glands, the contents of which obtain access to the general venous current or that in the pulmonary vein and its branches. The predominance of the infection in the lungs, in the liver, or in the area of the general circulation, depends upon the point of entrance of the bacilli into the blood stream. If a small artery furnish the point of infection the miliary eruption may be confined to the narrow limits of its distribution. The original focus may be at any point in the body where a breaking-down tuberculous deposit exists, but probably a majority of all cases originate from the respiratory tract. Three per cent. of all cases of pulmonary tuberculosis are believed to eventuate in general miliary tuberculosis.

The exciting cause may be almost anything that depresses the general resisting power, upon the one hand, such as chronic disease (cirrhosis of the liver, arteriosclerosis, nephritis, etc.), when miliary tuberculosis occurs as a terminal infection; or, upon the other, the advent of an acute catarrhal infection, such as measles, whooping cough, typhoid, etc., or of trauma or operation upon infected glands, which leads to sudden activity upon the part of a quiescent focus.

**General Infection.**—The most typical form of acute miliary tuberculosis is the general infection or typhoidal form, so-called from its resemblance clinically to enteric fever. Because of the lack of localizing manifestations, it is not strange that perhaps half the cases are at first suspected to be typhoid.

**SYMPTOMS.**—Often after a period of ill-defined lack of vigorous health, and especially in those with some recognized focus of

tuberculous infection, a serious failure occurs, quite abruptly at times, with irregular fever, rapid pulse, perhaps chilliness, profound prostration and headache. Anorexia and cough are frequent and constipation is the rule. Marked nervous symptoms shortly appear, delirium, somnolence and coma often following the restlessness and hyperesthesia of the earlier days. The fever may be continuously high, but rather tends to irregularity, remissions, even intermissions, and occasionally to the inverse type. The dry tongue and rapid pulse resemble those features of typhoid, but dichrotism is uncommon.

Respiratory phenomena are often the first ones to attract attention to the character of the infection. A slight bronchitis with bronchitic or fine crackling rales, and accompanied by dyspnea and cyanosis out of proportion to the physical signs, should lead to suspicion of miliary tuberculosis.

Herpes labialis, Cheyne-Stokes respiration and nose bleed may be noted. The spleen is commonly enlarged and may be palpable, but less often than in typhoid. Since diarrhea, meteorism, diazo-reaction and febrile albuminuria may be present, confusion with typhoid becomes almost excusable. A further cause of confusion is found in the discrete red papules occasionally seen in infants suffering from miliary tuberculosis. They are two or three millimeters in diameter, and upon histological examination of the lesions, Tileston found tubercle bacilli in 70 per cent. of the cases.

A slight leukocytosis is occasionally noted, and the bacilli may be detected in the blood stream or in the spinal fluid. At this time the eruption of a profuse crop of miliary tubercles in a given organ may change the aspect of the disease from that of a general toxemic condition to one of the more definitely localized types of miliary tuberculosis.

The course is likely to be less than a month, though a fatal result may occur in less than a week, or not until well into the second month.

**DIAGNOSIS.**—The signs and symptoms described, in one known to have a focus of tuberculosis, suffice to make the diagnosis extremely probable. The finding of miliary tubercles in the choroid, or of the bacilli in the blood or spinal fluid renders the diagnosis positive.

On the other hand, we must not give undue weight to the presence of bacilli in the sputum, urine, fecal discharges, etc., nor to a positive tuberculin reaction, since it is entirely possible that another infection complicates that by the *Bacillus tuberculosis*.

The differential diagnosis contemplates in nearly every instance the differentiation between miliary tuberculosis and typhoid fever, since the latter is the type of general infection most commonly and

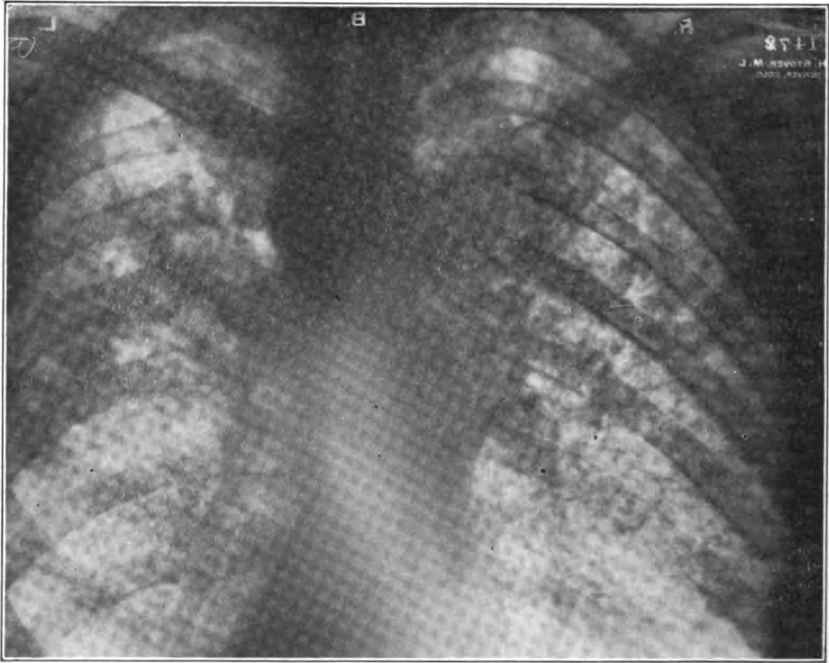


FIG. 18.—GENERAL MILIARY TUBERCULOSIS OF LUNGS. (Dr. G. H. Stover.)

most nearly resembling the former. A positive Widal reaction in one not recently infected with typhoid, or finding of the bacillus in the blood culture, may be regarded as absolutely decisive. On the other hand, typhoid patients, and especially children, may give a pronounced reaction to the tuberculin tests without materially affecting our judgment as based upon the general evidence, since latent tuberculosis is extremely common. Patients with miliary tuberculosis may show typhoid bacilli in the urine, but be merely carriers of the infection, not sufferers from it.

In general we may say that the presence of known tuberculous infection, infrequency of typhoidal infection in the community, irregularity of onset, irregular temperature curve, absence of diarrhea, nosebleed, stupid expression, dichrotic pulse, and typical rose spots, presence of marked cyanosis, and notably rapid respiration, all incline us toward the diagnosis of miliary tuberculosis. Most of the cases in my experience where doubt has existed have turned out to be of a tuberculous nature. It should not be forgotten that in the clinics of the best internists of the world the diagnosis of miliary tuberculosis is occasionally first made in the post-mortem room. Bonney expresses it well, however, when he states that failure in diagnosis more often results from neglect to utilize available means than from lack of proper data.

**Pulmonary Form.**—This is a result of infection from a breaking-down tuberculous focus, and occurs chiefly in those suffering from the pulmonary lesion. It not infrequently follows measles, whooping-cough, and typhoid fever. It may develop, however, in those in whom the infection has been so entirely latent as to have been unsuspected.

In one with well-developed pulmonary tuberculosis the onset may be so gradual as to be looked upon during the first days as an exacerbation of the usual symptoms from which the patient has been suffering. In others, the advent of a bronchitis, with chilliness, headache, and increasing expectoration, with "grazing" rales, if tubercles be developed profusely upon the pleura, or fine moist or bronchitic rales if they appear in the finer tubes, and with early development of the signs of a bronchopneumonia, characterize the infection. The sputum may be mucopurulent, rusty, or colored by fresh hemoptysis.

The general symptoms are those already described as characterizing a general infection. The cyanosis and rapid respiration are the most striking features in diagnosis. I have seen a respiratory rate of 80 per minute in an adult, with intense cyanosis.

**DIAGNOSIS.**—The diagnostic features of the pulmonary form of acute miliary tuberculosis have been sufficiently considered in the previous section, excepting that we may note that the presence of



tubercle bacilli in the sputum and the occurrence of dyspnea and cyanosis far beyond the degree to be expected from a consideration of the physical signs in the lungs should strongly suggest the pulmonary form.

**Meningeal Form (Tuberculous Meningitis).**—This type is said to occur as a primary disease, but the overwhelming majority of cases occur in those showing post-mortem either pulmonary tuberculosis or infection of the tracheobronchial or mesenteric glands. The cerebral meninges are chiefly involved, and notably at the base of the brain. The acute infectious diseases or trauma occasionally precede the development of meningeal tuberculosis. It is remarkable that the disease seems almost as likely to arise in one whose tuberculous process is believed to be wholly latent, as in one with advanced phthisis. A majority of the cases occur in children during the first four or five years of life. In Colorado the presence of great numbers of patients suffering with pulmonary tuberculosis explains the comparative frequency of tuberculous meningitis in adults. Out of 52 cases reported in 1905, 29 occurred after the twentieth year,\* but a similar proportion would perhaps be found in no other city.

**SYMPTOMS.**—As prodromata we may note fretfulness, irritability, loss of appetite and of weight, headache and change in disposition. Either suddenly or gradually more definite symptoms may appear—severe headache, vomiting, fever, photophobia, and retraction of the muscles of the neck. A convulsion may initiate the disease, and repeated convulsions may occur during its course.

The “hydrocephalic cry” is fairly distinctive. The headache is so intense that the child cries out, meanwhile holding the forehead or beating the head with hands and arms. The screaming may be almost constant. Restlessness, disturbed sleep and muscular twitching are common. Constipation and retracted abdomen are generally present. The fever is moderate and very irregular in character, and may be practically absent.

The respiration rate is not materially increased, unless the mil-

\* Hall and Hopkins: “Tuberculous Meningitis, with Report of 52 Cases,” *Jour. Nerv. and Mental Diseases*, April, 1906.

iary invasion involve the lungs. The pulse becomes slow as the disease advances, and its fairly constant irregularity constitutes one of the most characteristic features of the disease. The contracted pupils of the earlier days are likely to become dilated, disparity in size is frequent, and the pupillary reflex may be lost. As the rigidity of the muscles of the neck increases the head is retracted more and more, and the child's body may be raised from the bed by the hand placed under the occiput. Strabismus and ptosis are common. Kernig's sign is generally present and the knee-jerks disappear. Koplik found the Babinski reflex more commonly present in children over two years of age, than the Kernig sign.

As the disease advances, delirium, coma, convulsions, muscular spasms, monoplegia or hemiplegia, aphasia, blindness and deafness may appear. Patches of erythema, the tache cérébrale, herpes labialis, retracted abdomen, dry tongue, coma, involuntary discharges, fluttering pulse and finally death complete the melancholy picture.

Certain variations should be noted. The general hyperesthesia observed in cerebrospinal meningitis is occasionally present. Persistent vomiting or persistent hiccup may be noted. Early monoplegia, hemiplegia, or ophthalmoplegia may occur. Persistence in one position, usually a lateral one, with characteristic screaming when disturbed, is very suggestive of meningeal tuberculosis. The feet commonly turn outward as the patient lies upon his back.

In no other fatal disease is it so common to have periods of apparent improvement after apparently hopeless coma. I have seen both attendant and consultant discharged because of the refusal to change the diagnosis and the ultimate prognosis in the case of a baby which had lain comatose for three or four days, but was found sitting up in the crib playing with a doll, apparently convalescent at the time of the morning visit. Death in a few days confirmed the original diagnosis, as it has always done in similar cases which I have seen. In one case, a mechanic with a recognized pulmonary tuberculosis, after meningitis of three weeks' duration, in which an absolutely hopeless prognosis had been given, recovered so far from all meningeal signs and symptoms as to apply at the shops for his old

position. He relapsed the next day, with fatal outcome during the week.

Macewan's sign is present in more than half the cases—tympanic resonance upon percussion over the pterion with the child supported in the upright position. It signifies the presence of the acute internal hydrocephalus which formerly gave the name to this disease.

Lumbar puncture generally yields a turbid or hazy fluid, often under increased pressure. Tubercle bacilli may commonly be found if sought for with sufficient care (24 out of 27 of Koplik's cases). An increase of the total number of cellular elements, with over 30 per cent. of mononuclear cells, is commonly noted. A predominance of polymorphonuclear leukocytes suggests some other form of meningitis.

**DURATION.**—This varies from a few days to several months. Eskridge reported one case fatal after seven months' duration. The average time is probably three or four weeks.

**DIAGNOSIS.**—This must consider both the possible presence of meningitis and the variety of the disease. Nearly all the signs of meningitis may be present, in children especially, in the acute infectious diseases, when the later post mortem evidence points to a congestion of the meninges as the sole lesion. The diagnosis of "meningitis," in a considerable proportion of cases, should read "meningeal irritation," and not until signs of pressure upon the cerebral nerves or other definite localizing phenomena appear should the possibility of irritation from congestion and general toxemia be set aside.

In the presence of the signs and symptoms described above, lumbar puncture should be made. The finding of normal fluid suspends the positive diagnosis of meningitis for a time at least. The differential diagnosis between tuberculous meningitis, pneumococcic meningitis, the epidemic form, septic meningitis, etc., must depend ultimately upon the laboratory procedures. The presence of pneumonia or bronchopneumonia, or of a general streptococcic infection, or of the eruption and other signs noted under epidemic meningitis, will suggest careful examination for the respective forms of meningeal inflammation indicated. Middle ear disease, mastoid involvement,

thrombosis of the sinuses or other possible source of infection should be carefully sought for in case of doubt.

**PROGNOSIS.**—Death practically always occurs in any of the forms of acute miliary tuberculosis. The scattered cases of recovery from meningitis after absolute demonstration of tubercles in the choroid and of tubercle bacilli in the spinal fluid, and the reported finding of the evidence of healed meningeal tubercles post mortem are sufficient to prevent our ever giving an absolutely certain fatal prognosis, but do not justify the holding out of any material encouragement in a well-marked case.

### B. PULMONARY TUBERCULOSIS: CONSUMPTION

We shall consider the pulmonary form of tuberculosis under the following headings:

- (1) Acute pneumonic phthisis;
- (2) Acute bronchopneumonic phthisis;
- (3) Chronic ulcerative phthisis;
- (4) Fibroid phthisis.

(1) **Acute Pneumonic Phthisis.**—Acute pneumonic phthisis is more common in adult males than in other subjects, and so closely resembles lobar pneumonia in its onset that it is, in perhaps a majority of cases, at first mistaken for that disease. The chill, pain in the side, cough, expectoration, frequently rusty, high fever, rapid pulse, dyspnea, with the usual physical signs of acute pneumonia, are in fact the data upon which the diagnosis of the latter disease is commonly based. Either a single lobe, an entire lung, or an involvement similar to that in double pneumonia may be noted.

The course is that of acute pneumonia up to the time of the expected crisis, the failure of which may first excite suspicion. Even then a delayed resolution may appear more probable than the less common disease we are studying. Unless the examination of the sputum has shown tubercle bacilli, sometimes found even in the first week, or the previous family and personal history of the patient arouse our fears, it is likely that the erroneous diagnosis of acute pneumonia may be adhered to until an increasing expectoration, of-

ten greenish in color, sweats, hectic fever, loss of flesh, and general failure force themselves upon the attention. Cavity formation and the presence of elastic tissue in the sputum may now appear. The occasional presence of the pneumococcus in the sputum in the earlier days and sometimes later in association with the tubercle bacilli still further adds to the confusion. I have known the expectoration to reach a quart in amount during the 24 hours within 20 days of the onset.

Hemoptysis may occur, generally at the onset. Cyanosis and albuminuria are less frequent than we might expect them to be. The patient may die even as early as the sixth day, but commonly lives several weeks or two or three months. The severity of the process may become moderated, and the case pass over into the chronic form of phthisis, but few recover.

DIAGNOSIS.—This is ordinarily impossible until the finding of tubercle bacilli in abundance in the sputum, often with elastic tissue, coincident with a failure of resolution of lung involvement, with the fever and wasting mentioned, demonstrate that the case is not one of acute pneumonia. The latter disease is so frequent as a complication of chronic phthisis that the mere finding of the tubercle bacilli by no means suffices for a diagnosis of the acute pneumonic form of phthisis.

(2) **Acute Bronchopneumonic Phthisis.**—The bronchopneumonic form may occur in adults or in children, and as to diagnosis bears much the same relation to ordinary bronchopneumonia as the pneumonic form of phthisis bears to acute pneumonia. It is the form of tuberculosis (caseous bronchopneumonia), commonly known as galloping consumption.

Fraenkel mentions three forms of tuberculous bronchopneumonia. The first occurs as a secondary infection from aspiration of blood or secretions or both from a tuberculous focus, often from a cavity in the apex, and presents the physical signs of the ordinary bronchopneumonia. The second form presents small caseous foci surrounding the bronchi, with less-marked physical signs and perhaps without the presence of tubercle bacilli in the sputum. The third form occurs as a disseminated ulcerative process in those enfeebled by

chronic disease, such as diabetes and alcoholism, and in young children debilitated by attacks of measles, whooping-cough or influenza. Hemoptysis may be the cause of death, and general miliary invasion is not uncommon.

The onset is less acute than in the pneumonic form. Irregular chilly sensations, with fever, loss of appetite, failure in weight and strength, cough, expectoration, not often profuse, occasional hemoptysis, and perhaps pain in the chest characterize the earlier days of the illness. In the affected areas may be found moderate dulness and moist rales at the beginning, often modified later by the occurrence of more extensive deposits, by cavity formation, or by compensatory emphysema. Tubercle bacilli and elastic tissue are commonly present in the sputum at this time. In the severer types a typhoidal state supervenes. The lesions commonly spread until they merge into each other, and death occurs in the course of a few weeks.

**DIAGNOSIS.**—This ultimately depends upon the finding of the specific organism in the sputum, frequently with the evidence of destruction of lung tissue. The disease is to be suspected in children when a bronchopneumonia supervenes upon the catarrhal infectious diseases mentioned above, and in adults when a previous infection with tubercle bacilli is known. The early bronchopneumonia occurring as an aspiration process after phthisical hemoptysis may be wholly non-specific in character, but a diffuse tuberculous bronchopneumonia is not uncommon somewhat later after the invasion.

The disease commonly ends fatally in a few weeks, or, in children, even in a few days. It rarely passes into a chronic form of tuberculosis, and even then recovery is not to be expected.

**(3) Chronic Ulcerative Phthisis.**—Chronic ulcerative phthisis is by far the most frequent form of pulmonary tuberculosis, and is the form to which the term “consumption” is commonly applied. At first a local process, commonly just below the apex of one lung, the deposit of miliary tubercles, patches of caseous bronchopneumonia, and finally diffuse tuberculous infiltration, extends; and later softening and necrosis occur. By a process of ulceration cavities are formed, and the lung may become shrunken by the sclerotic process about the cavities and by the pleural thickening and adhesions over the spots

where the tuberculous process has reached the periphery of the lung. Bronchiectasis, shrinking of one side of the chest, displacement of the heart, compensatory emphysema, pleural effusion, tuberculous bronchial adenitis, fatty liver, amyloid degeneration of the liver, spleen, etc., laryngeal and other visceral involvement, chronic nephritis and general miliary tuberculosis may be successive features in the progress of the disease.

The beginning of the infection is commonly found just below the apex, posterolaterally, rather more frequently upon the right side. It extends downward in the upper lobe, and has a tendency to invade the opposite apex, the upper portion of the right middle lobe, or the apex of the lower lobe upon either side. While tuberculosis originating in the lower lobes is uncommon, this location alone should not weigh against the diagnosis.

Recovery is dependent upon the success of the sclerotic processes in their attempt to encapsulate the tuberculous focus, inspissation and final calcification signifying the victory.

**MODE OF ONSET.**—Tuberculosis of the lungs may start insidiously without a definitely fixed time of onset. It is more common for it to develop after a cold, ordinary bronchitis, influenzal attack, after measles, whooping-cough, typhoid or other disease in which a bronchial catarrh has been present, after pleurisy, recognized to be tuberculous in the great majority of cases, or after an initial hemoptysis. Bonney found that in nearly 20 per cent. of his cases the hemoptysis was the first signal of disease, although pathology teaches us that the tubercle bacillus has already initiated destructive changes before the bleeding occurs. It is not infrequent to find involvement of the larynx, or bronchial or cervical glands, or other organs preceding the development of the disease in the lung, although such involvement is commonly secondary.

The frequency of some form of exposure, giving rise to bronchitic changes, with eventual tuberculous infection, should be noted. The various occupations in which deleterious substances are inhaled, whether of organic nature, as dust in cotton mills, flour dust in flour mills, floor dust in the sweeping of public buildings, etc., or of inorganic nature, as in miners, grinders, etc., give rise to pneumocon-

iotic processes, but a secondary infection with tubercle bacilli is extremely common as the final cause of death.

Those working in over-heated and ill-ventilated rooms easily take cold, and many cases of pulmonary consumption may be dated from some such definite exposure. Several physicians from the natural gas regions who have been under my care for pulmonary tuberculosis have stated that the exposure in driving home at night in winter, especially after a confinement, from a house over-heated by the fuel mentioned, and commonly ill-ventilated, was the immediate cause of their illness.

It goes without saying that in all the types described, the infection with the tubercle bacillus from without, or a breaking out of a latent lesion within, constituted the essential step in the development of the pulmonary tuberculosis.

**SYMPTOMS.**—These are at first general in character, and consist of weakness, loss of appetite, anemia and digestive disturbances, and inability to do accustomed work without unusual fatigue. Loss of weight, gradually increasing cough, slight expectoration, a rise of a degree or two in temperature in the afternoon, especially after exertion, increased frequency of the pulse, night sweats, pain at one apex, flushed face during the time of pyrexia, often upon the affected side, dilated pupils and lowered blood pressure soon appear. Hoarseness at this time is more commonly due to a laryngeal catarrh than to the tuberculous infection of the larynx which so often complicates the later stages. Hemoptysis is so much more frequent in tuberculosis than in all other diseases combined, at least in this country, that its advent in any case, but especially in one presenting some of the foregoing symptoms, is of great diagnostic value. Its pulmonary rather than cardiac origin is easily established.

In the incipient cases tubercle bacilli may not be detected in the sputum, although they may be found even in the blood of the initial hemoptysis. Their presence or absence depends upon whether the disease process is open or closed, that is, upon whether the softening of a tuberculous lesion has permitted the bacilli to escape or not. Because of the fact that pulmonary tuberculosis may exist for months or even years before such softening occurs, the absence of tubercle ba-



cilli should not be regarded as weighing too seriously against the diagnosis.

**PHYSICAL SIGNS.—*Inspection.***—The patient is likely to be thin and to present the so-called phthisical chest, long and narrow, and with widened interspaces. Flattening at one apex, with lessened mobility, is frequently noted. Palpation reveals little in the earlier stages, unless it be the limitation of motion upon one side, sometimes better felt than seen. Later an increased fremitus, especially over solidified lung, or over pleuritic adhesions, may be manifest, and slight rigidity of the intercostal muscles in certain cases. Very light percussion may show deficient resonance and higher pitch in the affected apex, especially if it be practiced while the lungs are distended. The normal slight lack of vesicular resonance at the right apex must be borne in mind.

Auscultation ordinarily reveals the first decisive evidence of the infection to be obtained by physical examination, for all the evidence heretofore mentioned is suggestive only. The preponderating importance of fine moist rales upon one side, often appearing after cough practiced at the end of expiration when otherwise inaudible, should be more widely recognized. The suspicious regions of the chest should be examined with the stethoscope before any deep breathing is permitted, for the reason that a few suspicious clicks, which may, early in the development of the pulmonary tuberculosis, constitute the most valuable physical evidence of the disease, may disappear upon repeated distension of the lungs by deep respiratory movements. In case of doubt the suspicious region should be examined by auscultation at the very beginning of the second examination. Unfortunately in many cases we must for weeks be content with far less dependable auscultatory evidence—prolonged and roughened breathing, cog-wheeled or interrupted respiration, a bronchovesicular element in the respiratory murmur, in regions where the sound should be purely vesicular, or rarely a pleuritic friction rub. The whispered and spoken voice sounds are increased.

A subclavian systolic murmur, so often mentioned, is not a sign of incipient tuberculosis, but of changes ordinarily produced by contraction of lung or pleural adhesions, distorting or compressing the vessels.

The physical signs of early tuberculosis are not uncommonly slight and rather indefinite. They must be at least fairly constantly demonstrable, and accompanied by some symptoms of the disease, to be taken into account. In case of doubt, further examination is imperative, and will generally settle the question.

The immense importance of immediate attention to the signs of an early tuberculous infection can hardly be overstated. The tendency to consider localized rales as indicating only a simple bronchitis, or more especially "localized" bronchitis, is not only in defiance of all that we know of the pathology of lung disease and of its detection by auscultation, but it is most deleterious to the patient's welfare, by leading to delay in taking the steps which lead to recovery from tuberculosis, at a time when they are of the utmost value. The sputum should be obtained, often despite the patient's assurance that there is not sufficient for examination. In the watery or glairy fluid, with more opaque sediment floating near the bottom, the bacilli may often be detected. Their presence establishes the diagnosis, but their absence at this stage proves nothing, since many of the cases are "closed." In many specimens of sputa, grippe bacilli, pneumococci and streptococci may be noted, and should lead to repeated examination for the more definitely diagnostic bacilli of tuberculosis.

If the diagnosis be not definitely established by means of the procedures thus far described, further observation is necessary. The temperature should be carefully noted for several days or weeks, a slight evening rise being of great significance.

**TUBERCULIN REACTION.**—If used under proper conditions the tuberculin tests have proved so free from danger that one of them should now be applied if doubt still exists. The methods of application and the interpretation of the results are far more satisfactory than they were two or three years ago.

The tuberculin reaction, within certain limitations, is of much value, but practically only in association with other methods of diagnosis. I quote from Dr. S. Simon, who has had very extensive experience with its use in the National Jewish Hospital for Consumptives.

"The difficulties surrounding the diagnosis of early tuberculosis

by means of physical examination of the chest, and the inability to find tubercle bacilli in the sputum in a certain percentage of tuberculous patients, have led to the use of tuberculin by subcutaneous injection. This is used upon the theory that the presence of tuberculosis in the body produces a change in the cells of the body which leads to the formation of a substance designated as tubercular lysin (Wolf-Eisner), which is capable of responding to tuberculin applied either subcutaneously or dermally, producing a general or local reaction. Koch recommended the subcutaneous injection of old tuberculin, an initial dose of .1 to 1.0 mg. being given; if no general reaction, as indicated by rise in temperature of one or two degrees, accompanied with headache and general malaise, occurs, a second dose of 2 to 5 mg. is given two days later; and if then no reaction occurs, two days later a third dose of 5 to 10 mg. If there be still no reaction, the patient is to be regarded as free of tuberculosis. Many authorities believe this method to be accompanied with some danger of lighting up an old tuberculous focus which may have been quiescent; for this reason very few physicians use this method.

"Various modifications of this method are now used; Lowenstein, in order to avoid the possible error of a quantitative non-specific reaction in hypersensitive non-tuberculous individuals by the use of increasing amounts of tuberculin, employs within an interval of twelve days four doses all alike, and amounting to 2-10 of a mg., of tuberculin. This has also been modified by employing first a small dose, then a second larger dose, then a third smaller dose, within a period of eight days.

"A test simultaneously advocated by Calmette and Wolf-Eisner consists in dropping one or two minims of  $\frac{1}{2}$  or 1 per cent. solution of glycerine-free, old tuberculin into the conjunctival sac of the eye; if no inflammatory reaction follows within twenty-four hours, the patient is considered free from tuberculosis. It is claimed that a positive reaction indicates the existence of an active tuberculous lesion (Wolf-Eisner). Where the first application results negatively, a second application of tuberculin, 1 per cent. to 4 per cent., is made to the conjunctiva of the other eye a few days later. Many cases of severe inflammatory reaction were reported from its use, and in some

instances permanent injury to the eye. For this reason this reaction is employed by but comparatively few physicians. It should never be used in the presence of any diseased condition of the eye.

"The test most commonly used at the present time is that introduced by Von Pirquet; this consists in the use of 25 per cent. strength of old tuberculin prepared by taking one part of Koch's old tuberculin and adding three parts of sterile normal salt solution. The technique is as follows: After cleansing a small area of the arm with alcohol or ether, a drop of control (which consists of one part 5 per cent. carbolic in glycerin, and three parts sterile normal salt solution) is inoculated with a spear—or chisel-shaped scarifier; one inch above and below the control a drop of the 25 per cent. O.T. is similarly inoculated, care being taken that the scarification be only sufficient to abrade the skin and not to produce a flow of blood. Instead of a 25 per cent. solution of tuberculin, many now use tuberculin in full strength.

"An areola of redness 5mm. or more in diameter appears in from twenty-four to forty-eight hours around the points inoculated with tuberculin, if a positive reaction results, but no such redness around the control; the redness varies in intensity; a negative result is indicated by a similarity of appearance of both tuberculin scarifications to the control.

"Another method frequently used is Moro's percutaneous test; this consists in the use of equal parts of old tuberculin (Koch) and lanolin. The latter is heated to 100°F. and the tuberculin thoroughly mixed with it in a mortar. A quantity of this ointment about the size of a pea is rubbed into the skin of the abdomen or arm, the site first having been cleansed with soap and water followed by alcohol or ether. A positive reaction is indicated by an herpetic-like eruption in a field of redness, corresponding to the area in which the inunction was given. Several degrees of reaction are obtained, varying from a few discrete papules to numerous papules in an area of redness of greater or less intensity.

"A fourth local test should be mentioned, known as Lignier's, which consists of rubbing into a small cleansed area of the arm a few drops of undiluted tuberculin. The results of this test are similar

to those obtained with Moro's ointment. Still another local test is known as Hamburger's 'Stich reaction.' This consists in the intradermic injection of a small quantity, 0.1 c.c. of 1 per cent. solution; at the site of the injection, there occurs after forty-eight hours an infiltration varying in size and intensity. In order to exclude the existence of hypersensitiveness to tuberculin, he precedes his Stich reaction by the cutaneous reaction with undiluted tuberculin.

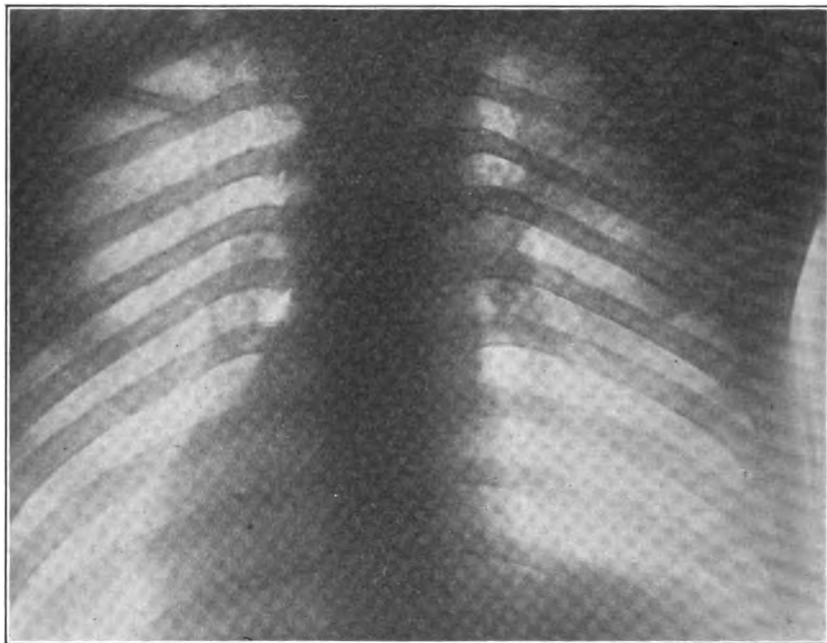


FIG. 19.—RÖNTGENOGRAM SHOWING COURSE OF EXTENSION OF TUBERCULOSIS FROM ROOT TO APEX, WHERE PHYSICAL SIGNS INDICATED INCIPIENCY. (Dr. G. H. Stover.)

“Experience has shown that these various tuberculin tests are of some value in the diagnosis of tuberculosis when properly estimated. A positive reaction occurring in children under three or four years of age, who are in poor health, indicates the existence of an active tuberculous lesion somewhere in the body. In older children a positive reaction, together with other symptoms of tuberculosis, would be of value as pointing towards an active tuberculous lesion.

"In adults, not much reliance can be placed upon these tests alone as diagnostic aids as to the existence of clinical tuberculosis. When one recalls the fact that nearly all mankind, and one may safely say all city dwellers, have at some time in their lives come in contact with the tubercle bacillus, and that the tuberculin test is positive in all cases where a tuberculous infection has ever occurred, its value in the diagnosis of clinical tuberculosis cannot be very great. Nevertheless, a history of gradual loss of weight, anorexia, a slight after-

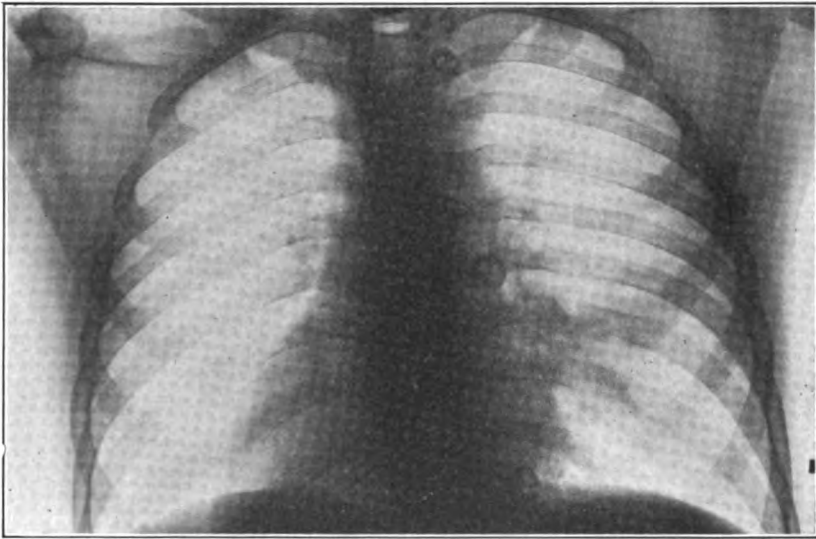


FIG. 20.—CASE OF TRANSPOSITION OF VISCERA. Röntgenogram shows marked tuberculous involvement of lymphatic tissues at the root of the lung on the left side. The child's failing health had been attributed to disease of the accessory nasal cavities. (Dr. G. H. Stover.)

noon rise of temperature, a possible exposure to tuberculous infection with a positive tuberculin integumental reaction, even where the physical findings are negative, should be regarded as pointing to the existence of active tuberculosis.

"Far advanced cases of tuberculosis often give a negative tuberculin reaction for the reason that the body cells are so overwhelmed by the toxins of the tubercle bacillus that they are incapable of forming the 'tubercular lysin.' The same is true of tuberculous meningitis in children.

"Summarizing, one may say that the tuberculin test is of considerable value as a diagnostic agent in children under three years. In older children and in adults it has in itself little value as indicating the existence of clinical tuberculosis, unless judged in proper perspective with other symptoms."

**RÖNTGEN-RAY EXAMINATION.**—In the early stages of tuberculosis of the lungs little that is of positive significance is to be detected by this means. The slight shadow over the affected region, and the limitation of the lung movements, are, like the signs obtained at this stage by inspection, palpation, etc., suggestive rather than of positive diagnostic value. In the later stages and especially in the face of certain complications the X-ray is of great value. "Groups of miliary deposits give a mottled image of small grain, as compared to the larger, denser areas due to inflamed or calcified glands or focal pneumonitis." (Stover.)

For the detection of incipient tuberculosis in the lungs by physical signs, it is necessary that the lung tissue be practically normal to start with. The supervention of a tuberculous infection upon the fibroid induration resulting from the inhalation of dust, etc., or in cases of chronic bronchitis and emphysema, or in old cases of pleurisy, must be detected rather by examination of sputum, changes in the temperature curve, tuberculin reaction, etc., than by physical signs.

### *More Advanced Tuberculosis*

**SYMPTOMS.**—As the pathological changes in the lung advance, cough becomes more annoying, expectoration more profuse, the sputum more purulent, fever more marked and regular, the pulse more rapid and the emaciation more noticeable. Sweating during sleep becomes troublesome. Even with marked physical signs dyspnea may be rather less conspicuous than we might anticipate. The extension of the infiltration leads to an involvement of the pleura, and pain, usually at one apex or in the region of the nipple, is not uncommon. It may extend to the base of the lung. The anorexia and especially the repulsion to the taking of fatty food is more marked.

The importance of a feeble digestive power in giving an opportunity, through diminished tissue resistance, for the lodgment and growth of the bacilli of tuberculosis in the lung should not be overlooked.

As the disease advances, menstruation ceases, unless it has already disappeared, as is often the case in anemic young girls in the incipient stages. The idea that amenorrhea is the cause rather than effect of the disease still lingers in the minds of many mothers, and must be dispelled.

**INSPECTION.**—As the disease progresses we may note unhealthy color and greater emaciation. Inspection of the chest shows diminished respiratory movement over the region involved, especially if it be in one apex in front, over or under the clavicle. The retraction of this region is a feature of the more advanced disease. Patches of *tinea versicolor* are frequently noted over the front of the chest at this time, the lessened tissue resistance being favorable to the growth of the parasite.

**PERCUSSION.**—Moderate dulness is to be found over the area involved. The need of adjustment of the force to be used in percussion to the conditions present should be mentioned, for over-forcible percussion strokes fail to reveal minor changes in the lung beneath. The regions about the clavicle should be examined with especial care, for at this time dulness and higher pitch of the percussion note almost infallibly denote the area affected. With the arms folded in such a way as to separate the scapulae widely, dulness may also be found in the upper central dorsal regions or in the suprascapular fossae. The tympanitic note to be found in some cases where the consolidation beneath dampens the vibrations of the overlying lung tissue, and especially when slight compensatory emphysema is present, should be noted. Tympany may also be found over a superficial cavity. Flatness indicates either complete solidification of lung, great thickening of the pleura, or pleural exudate, but pertains more especially to the advanced form of tuberculosis than to the form we are considering.

**AUSCULTATION.**—Auscultation yields in advancing tuberculosis, as in the incipient form, the most trustworthy evidence to be obtained by physical examination. Moist rales, generally in the upper



portion of the lung, and often upon one side only, are practically constant at this time, though they may not be persistent throughout an examination. They are most easily found at the end of a fairly deep inspiration, which may be taken in doubtful cases at the end of a full expiration followed by cough. If the bronchitic exudate be present in even a moderate degree, however, the moist rales will be heard constantly over the infected area, during both inspiration and expiration, and without the need of coughing to bring them out. If the lung tissue around the tubules involved be to some extent consolidated, the rales are high pitched and have the quality of "nearness." Bronchial respiration is not commonly a marked feature of moderately advanced phthisis.

Occasionally sonorous or sibilant rales may be present, when fine rales cannot be detected. Their presence is of some value as to the diagnosis of phthisis if they be confined to one region of the chest. The essence of the diagnosis in pulmonary tuberculosis, so far as physical examination goes, may be stated to lie in the detection of changes indicating a persisting alteration in the structure of the lung and the patency of the finer bronchial tubes over a localized area, especially if this be in an apex. The exact character of the sounds indicating these changes may be of less import than the fact that the unilateral changes are present.

Intensification of the whispered voice, with moderate elevation of the pitch, increased vocal resonance and fremitus, bronchovesicular respiration and frequently the presence of wavy respiration, and the quality of roughness, are also frequently to be noted. It is necessary to exclude, of course, obvious causes of unilateral and persistent changes in the lung and pleura, such as occur after empyema, pleurisy with effusion, bronchiectasis, etc., but this procedure commonly presents no difficulties.

#### *Advanced Pulmonary Tuberculosis*

With the advent of mixed infection at the site of the pulmonary lesions, the fever becomes remittent or intermittent. The hectic fever of advanced phthisis is associated with destructive lesions in the lung

and cavity formation, and is essentially septic in character. A profuse colliquative sweat accompanies the morning fall of temperature. The pulse now becomes rapid, soft, compressible and frequently dichrotic, and the blood pressure is lowered. Diarrhea, edema of the legs and the peculiar *spes phthisica* are to be noted in many cases. Delirium may occur.

**INSPECTION.**—Inspection now reveals features from which alone, practically speaking, the diagnosis may be made. The pallor, emaciation, obvious dyspnea, perhaps cyanosis, and frequently edema of the feet, may attract the attention of the traveller upon almost any train bound for the regions where climatic or sanatorium treatment is to be obtained. Upon stripping the patient the emaciation permits the chest conditions to be much more readily noted than in the incipency of the disease. The prominent ribs and clavicles show by contrast the retraction of the lung beneath. The heart is commonly displaced to some extent, and shows more of a pulsating area than normal because of its being uncovered through retraction of the lung border normally covering it in part. This is more noticeable upon the left side than in involvement of the right lung, since the tendency in the latter case is for the heart to retreat beneath the sternum.

Failure of expansion of one lung, sinking of one shoulder, clubbed fingers and toes, cyanosis of the nails and a tendency to fullness in the hepatic region because of fatty or amyloid changes in the liver, dryness of the hair and a patchy, yellowish discoloration of the skin, may all be noted.

**PALPATION.**—This may show deficient expansion upon one side, increased area and force of the cardiac pulsations (already mentioned under inspection), increase of vocal fremitus over areas of consolidation, where air is still freely admitted, or decrease where, from pleural effusion or other cause, the lung becomes impervious, and possibly a friction rub over an area of pleuritic involvement. Enlarged tuberculous glands may be found.

**PERCUSSION.**—At this stage marked changes in the physical conditions within the chest lead to corresponding changes in the percussion note over the area involved. Marked dulness, even approaching flatness, may be present, most frequently in the upper chest.

Consideration must be given to the possibility that the flatness, when present, may be due to effusion. The diminution of the normal vesicular resonance is never uniform over the chest, different regions offering marked variations as to lessened intensity, changes in quality and elevation in pitch. Much judgment is necessary in the adjustment of the force of the percussion stroke in accordance with the

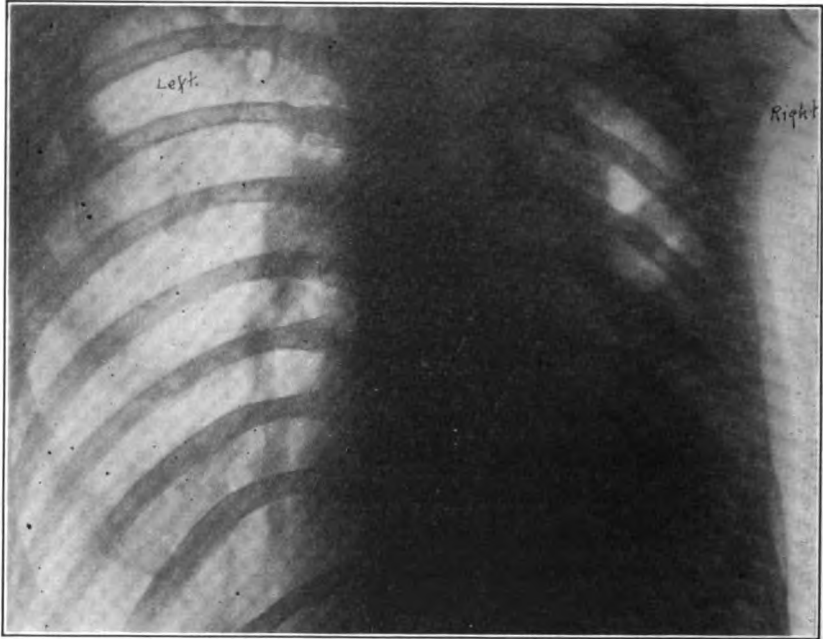


FIG. 21.—CAVITIES IN CONTRACTED, CONSOLIDATED RIGHT LUNG. Right chest collapsed. Considerable left-sided compensatory emphysema. Heart drawn and pushed completely to the right. Postero-anterior view. (Dr. G. H. Stover.)

thickness of the chest wall and the extent and depth of the area of lung tissue involved.

Tympany may be obtained over regions where small areas of solidification are scattered through the lung tissue in which compensatory emphysematous changes have occurred. Over cavities near the surface and not filled at the time of examination the same sign may be found. If the cavity be filled with secretion or be deep in the lung, or be relatively small, the percussion signs may not be decisive.

Cracked-pot resonance is often observable over superficial cavities. Wintrich's sign, Friedreich's sign and Gerhard's sign may be present.

**AUSCULTATION.**—As the destructive lesions in the lung advance, the roughened, cog-wheel, or bronchovesicular respiration, with a few moist rales is likely to be replaced by more decidedly abnormal

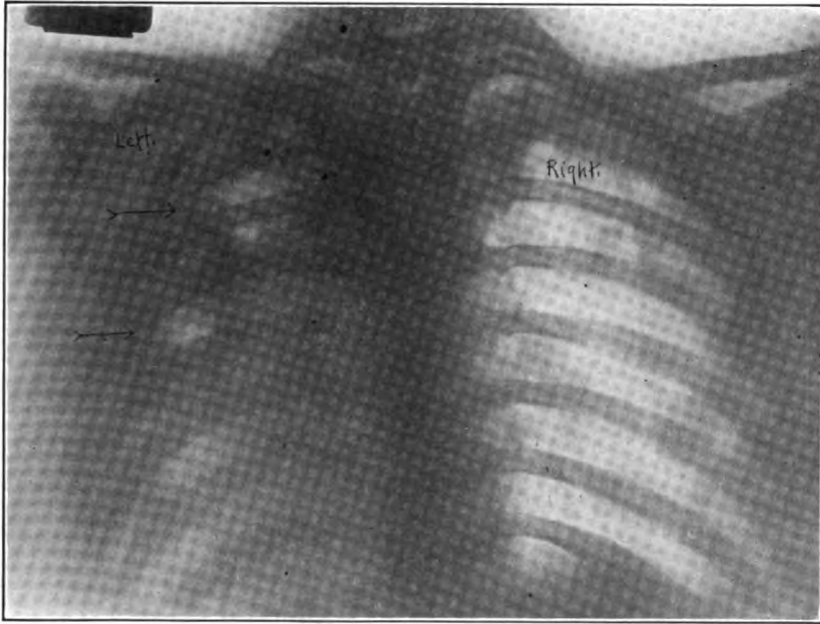


FIG. 22.—CAVITIES IN CONSOLIDATED, CONTRACTED LEFT LUNG. Heart drawn and pushed to left. (Dr. G. H. Stover.)

auscultatory phenomena. Constant moist rales, much coarser than those of the earlier stages, and varying much in size, abundance and "nearness," and in quality, are present, in a majority of cases.

As the softening of the lung advances the rales become more striking in character until, finally, with the advent of definite cavity formation, gurgling rales appear. If advanced solidification occurs, the rales are apparently extremely superficial, and are loud and harsh in tone. Vocal fremitus, sharp bronchial respiration, bronchophony and whispering pectoriloquy are found, the latter suggesting search

for vomicae. Cavernous respiration may be detected if the cavities be large and superficial.

Pleuritic friction is a frequent phenomenon in advanced tuberculosis, and may be heard over the extending infection in the lung, and, with rather surprising frequency, over the less involved lung. Unusually clear transmission of the heart sounds suggests the likelihood that solidified lung tissue may be responsible for it. The increased obstruction to the pulmonary circulation frequently causes the pulmonary second sound to be accentuated. In cases of involvement of the left apex, the retraction of the area involved may so uncover the base of the heart upon the left that this accentuation may appear extremely marked.

Physical signs, apart from those described in the lung, are of some importance in advanced tuberculosis. Myoidema is suggestive of over-excitability of the muscles of the chest, or of the nerves supplying them, and should lead to a careful physical examination of the lungs, although of little specific diagnostic value. Clubbed fingers occur in a majority of the cases of advanced phthisis. Hypertrophic pulmonary osteoarthropathy is rare, but of much significance when present.

**COURSE OF THE DISEASE.**—Pulmonary tuberculosis may be arrested, especially if early diagnosis and intelligent treatment have been attained before any marked physical signs have been found, and toward the accomplishment of such a result every effort should be made. Complete arrest may well be permanent if the patient live an intelligent life. Unfortunately the majority of cases progress slowly from one stage to another, and eventually reach the stage of chronic ulceration. The duration may be anywhere from a few months to many years, a great majority falling within the limits of two to eight years. I have known a woman to raise a family of six children to adult age while signs of chronic tuberculosis persisted in her lung, several of these children becoming infected. The occurrence of partial arrest with subsequent outbreak and rapid advance of the disease is too common. Many cases of advanced phthisis die from the various complications of the disease, especially from pneumonia, hemorrhage, pneumothorax, amyloid disease, nephritis, laryngeal

involvement, meningitis, etc. Pope's tables show that 35 per cent. of all the progressive cases are dead within six months, 50 per cent. within one year, 91 per cent. within four years, while 3 per cent. or 4 per cent. live from ten to forty years. The disease is more acute in the young and commonly less acute in the aged than in adults of middle life.

*Complications.*—Hemoptysis is the most frequent one, occurring at some time during the course in about three-fourths of the cases. Extension of the tuberculous process to the larynx and to various viscera is extremely common. Tuberculous meningitis is not infrequent. Acute lobar pneumonia is a grave and not unusual complication of phthisis. If the pathognomonic sputum and bacterial evidence be wanting the diagnosis may be difficult in a patient seen for the first time.

Abscess of lung, gangrene and bronchiectasis may occur. Pleurisy in its various forms has been considered. Pneumothorax is found in about 5 per cent. of autopsies, but is less frequently detected clinically. Serous effusion may accompany it, but pyopneumothorax is more common. Cases of pneumothorax rarely recover.

Amyloid visceral disease follows especially in the cases with long continued and profuse purulent expectoration, especially with cavity formation. Peripheral neuritis is rare. Notable displacement of the heart is frequent in the chronic types of tuberculosis.

Addison's disease is not infrequent as a complication of pulmonary tuberculosis.

The extension of the tuberculous infection to other organs has already received attention. The kidneys become infected in about 8 per cent. of the cases.

*DIAGNOSIS.*—In general, the diagnostic features have been considered under the different stages of the disease. The family and personal history of the patient; any depressing conditions in connection with his work, place of residence, state of nutrition, etc.; the opportunities he may have had for the acquirement of the infection; previous diseases favoring the development of pulmonary tuberculosis, notably whooping-cough, measles, influenza, pneumonia, pleurisy and typhoid fever; the occurrence, perhaps remotely, of hemopty-

sis, and many other factors must be considered. The association of fever, cough and loss of weight is extremely suspicious. Rapidity of the pulse rate, exhaustion upon slight exertion, general weakness and slight anemia are suggestive. The finding of almost any departure from normal in the physical signs at one apex, with any group of the above mentioned symptoms, is confirmatory. Persisting moist or crackling rales in one apex should lead to a presumptive diagnosis of tuberculosis, and even the failure to find the bacteriological proof of its existence does not suffice to overthrow the presumption unless the signs are amply explained in some other way, or the microscopical examination of the sputum demonstrates that some other organism is present. It should be recognized that the streptococcus, influenza bacillus, pneumococcus and other organisms may be found in the sputum when the physical signs lead us to expect the presence of the tubercle bacillus. These cases should rather be suspected of being closed tuberculous cases with mixed infection than regarded as instances of "streptococcic phthisis," etc., for the clinical course and the eventual pathological findings justify this attitude. Even without autopsy, the death of a patient with chronic lung disease who has never presented microscopical proof of tuberculous infection, from a characteristic tuberculous meningitis, may serve to convince us of the underlying cause of the disease. In general we may say that pulmonary tuberculosis is so frequent that it is safer, both from the standpoint of correct diagnosis and out of consideration for the welfare of the patient to regard all doubtful cases as phthisis and treat them accordingly, since absolutely positive scientific diagnosis is less of a desideratum than the recovery of the patient, even though this occur without the demonstration of the character of his affection. This remark is imperative in view of the multitude of cases permitted to drift along, and often to continue at work, because, for example, the tubercle bacillus cannot be found in the sputum. If the patient has definite symptoms and the signs of a localized lung infection it is time to treat him as if he were tuberculous, if he is to be saved.

The demonstration of tubercle bacilli in the feces may speak for the presence of a tuberculous enteritis, but this finding is common

in those suffering from the pulmonary infection (40 per cent. to 60 per cent.). In the urine they signify a local lesion in the genito-urinary tract. The tubercle bacillus is occasionally found in the circulating blood, but in such cases the diagnosis could scarcely be doubtful even without this finding. The proof of the tuberculous nature of a discharge from the ear adds to the probability that an accompanying lung infection is of tuberculous nature. No assistance has been given us thus far in the way of serodiagnosis.

**DIFFERENTIAL DIAGNOSIS.**—In the absence of tubercle bacilli in the sputum the differential diagnosis may be difficult. A bronchitis may be limited to the apices, though probably never to one apex, but such a condition is not associated with the symptoms of pulmonary tuberculosis, nor with dulness upon percussion. Asthma, chronic bronchitis, bronchiectasis, unresolved pneumonia and other lung affections must be differentiated by their characteristic features. (See the respective headings.)

Much difficulty is often encountered in differentiating miner's phthisis, stone cutter's phthisis, and similar processes noted in connection with other occupations. A chronic bronchitis of irritative origin lies at the base of the pathological lung changes, with emphysema, infiltration of the bronchial glands, contractile fibroid changes, secondary phenomena in the pulmonary circulation, and not infrequently secondary infection with the tubercle bacillus. The history as to occupation and exposure is of vital importance in the diagnosis. Even the finding of the tubercle bacillus does not at all overthrow the diagnosis of pneumokoniosis, since it is so commonly a secondary invader.

Certain diseases causing long continued fever may be suggestive of pulmonary tuberculosis, notably subacute endocarditis, Hodgkin's disease, with little accessible glandular enlargement, Grave's disease, when the goiter is not especially obvious, tuberculosis of the kidney, pernicious anemia, syphilis and malaria. Careful examination should prevent error. In lung infection by the anthrax bacillus, the ray-fungus, the streptothrix, cladothrix, aspergillus, the lung fluke, and in psittacosis, the laboratory must be relied upon for the diagnosis.

Hemoptysis is occasionally assumed to indicate the existence of



tuberculosis without proper investigation as to the cause. I have seen several cases of advanced mitral stenosis sent to Colorado as supposed cases of tuberculosis because of repeated hemorrhages. In one of these repeated attacks of acute pulmonary edema occurred, with the characteristic pink fluid expectorated in great quantities. Bleeding from dilated veins at the root of the tongue or in the pharynx has been the explanation, given me by Dr. Robert Levy, of several cases of supposed hemoptysis in which I could find no pulmonary cause. A rather profuse hemorrhage occurred in the case of a physician from the strain of severe vomiting in an attack of migraine. His good health for nearly twenty years following the attack justifies the exclusion of tuberculosis.

PROGNOSIS.—Under ideal conditions as to the earliest possible diagnosis and the best treatment, probably 90 per cent. of the cases of pulmonary tuberculosis are curable. This statement is fortified by the post mortem findings as to healed tuberculosis, for we know that without diagnosis or treatment a great number, perhaps a majority of all infected with the bacillus, recover.

Family predisposition is of immense importance in prognosis. I have cared for several individuals who were the sole survivors of considerable families, all of whom had died of consumption. No such patient has recovered. I have noted but 10 per cent. of recoveries amongst 30 patients who have contracted pulmonary tuberculosis in Colorado. Those who succumb to infection in a region so favorable for the avoidance of it, and even for recovery after infection, have too little resistance to withstand it. None of the patients returning from our tropical possessions with tuberculosis has recovered under my observation. The same lack of tissue resistance which makes the venereal diseases so formidable in this class of patients seems to be operative in those infected with tuberculosis.

Youth and age are unfavorable. Bonney finds the outlook decidedly better in the female, and in this I concur. The Indian, the negro and the mulatto offer less resistance than the Caucasian. Favorable out-of-door occupation is a deciding factor as to recovery in many cases, and naturally the ability to seek the best climatic and other conditions for cure are to be considered.

Pregnancy is distinctly unfavorable, and marriage is often followed by sharp advance of the lesions. A relatively slow pulse, good digestion, well-balanced mental attitude and persistence in following the best advice are of the greatest help toward recovery.

**(4) Fibroid Phthisis.**—This is the term applied to the cases of chronic pulmonary tuberculosis in which the caseation and ulceration are overshadowed by the development of fibrous tissue. It is often a sequel to a tuberculous pleurisy, but may follow the other forms of pulmonary phthisis.

It is characterized by its long course, not infrequently reaching 15 or 20 years, by the marked shrinking of the affected lung, with deformity of the chest; by great displacement of the heart in many cases; by relatively slight fever; small amount of expectoration, frequently not purulent in character; and the slowly developing changes in the pulmonary circulation. Thickening of the pleura, enlargement of the bronchial glands, bronchiectasis, marked clubbing of the fingers, and progressive emaciation are features of the disease.

**SYMPTOMS.**—Chronic cough, especially on arising, slight expectoration, excepting in the cases with marked cavity formation or bronchiectasis, very moderate fever, when any rise of temperature is noted, marked dyspnea, cyanosis and progressive failure in weight and strength are noted.

**PHYSICAL SIGNS.**—Those relating to the lung are such as are associated with the thickening of the pleura, replacement of alveolar tissue by the fibrous growth, cavity formation, and bronchiectasis. The bronchial breathing, diminishing vesicular sounds and absence or rarity of rales in parts not affected by cavity formation or dilatation of the bronchi are notable. Hemorrhages are often frequent and severe.

Many interesting signs relate to the heart. It is commonly uncovered by the retraction of the affected lung, so that its beat is visible over a large space. I have seen it above and to the outer side of the right nipple. Hypertrophy of the right side of the heart and extreme pulmonic accentuation are common. Relative leakage at the pulmonary orifice, indicated by an inconstant, soft, diastolic murmur transmitted toward the apex, and without any of the fea-

tures of aortic regurgitation, is more frequently noted in this disease than in any other unless it be extreme mitral stenosis. I have noted it in several instances.

Many of the patients die of sudden profuse hemoptysis, others of gradually developing heart failure. There is danger of amyloid disease or of generalized tuberculous infection.

**DIAGNOSIS.**—This depends upon the recognition of the symptoms and signs described. The differential diagnosis relates to the decision

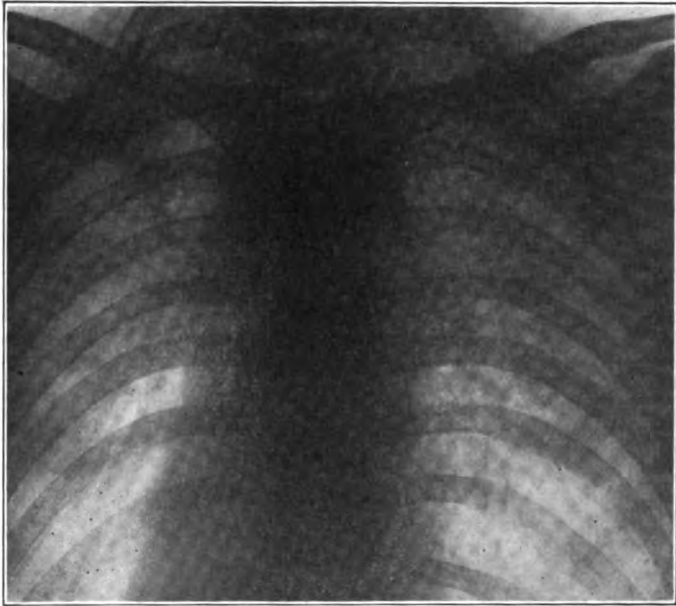


FIG. 23.—WIDE-SPREAD CHRONIC FIBROID PHTHISIS. Aorta displaced by contraction of fibrosing tissue. (Dr. G. H. Stover.)

as to whether a fibroid lung condition is of tuberculous origin or not. The presence of tubercle bacilli in the sputum or of tuberculous complications is decisive, but these points may be lacking. The tuberculin test may be applied.

**PROGNOSIS.**—Fibroid phthisis practically never results in complete recovery, but its mildness and chronicity are so marked that many individuals carry on successfully half a life's work before succumbing to the disease.

The following schema was adopted by the National Association for the Study and Prevention of Tuberculosis in 1905:

OFFICIAL CLASSIFICATION IN PULMONARY TUBERCULOSIS

INCIPIENT (favorable)...	Slight initial lesion in the form of infiltration limited to the apex or a small part of one lobe.
	No tuberculous complications. Slight or no constitutional symptoms (particularly including gastric or intestinal disturbance or rapid loss of weight).
MODERATELY ADVANCED...	Slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours, especially after rest.
	Expectoration usually small in amount or absent. Tubercle bacilli may be present or absent.
FAR ADVANCED.....	No marked impairment of function either local or constitutional.
	Localized consolidation moderate in extent with little or no evidence of destruction of tissue; Or disseminated fibroid deposits.
ACUTE MILIARY TUBERCULOSIS .....	No serious complications.
	Marked impairment of function, local or constitutional.
UNIMPROVED .....	Localized consolidation intense;
	Or disseminated areas of softening;
IMPROVED .....	Or serious complications.
	All essential signs and symptoms unabated or increased.
ARRESTED .....	Constitutional symptoms lessened or entirely absent; physical signs improved or changed; cough and expectoration with bacilli usually present.
	Absence of all constitutional symptoms; expectoration and bacilli may or may not be present; physical signs stationary or retrogressive; the foregoing conditions to have existed for at least two months.
APPARENTLY CURED.....	All constitutional symptoms and expectoration with bacilli absent for a period of three months; the physical signs to be those of a healed lesion.
	All constitutional symptoms and expectoration with bacilli absent for a period of two years under ordinary conditions of life.
CURED .....	

## C. GLANDULAR TUBERCULOSIS

**Definition.**—The term *scrofula* was formerly applied to tuberculosis of the cervical lymph nodes. This form is exceedingly common in children. Probably involvement of the tracheobronchial glands is equally common in youth, and in adults with pulmonary tuberculosis a secondary invasion of the latter glands is often found post mortem. *Tabes mesenterica* is more frequent in children.

The infection in the cervical glands is derived from the tonsils, adenoid vegetations, and from the mucous membranes of the upper respiratory tract; that of the tracheobronchial glands comes from the lungs and the bronchi, and is especially common in children after the catarrhal respiratory diseases. The involvement of the mesenteric glands is seen in association with intestinal tuberculosis, and often originates in the diarrheal disturbances of infancy. Behring believes that the digestive tract furnishes the source of infection for the bronchial glands in many instances.

The infection in the glands often becomes quiescent, and calcification may take place. The frequency with which the supposedly latent glandular infection gives rise to meningeal or general miliary tuberculosis should not be overlooked.

**Cervical Adenitis.**—The submaxillary glands are commonly the earliest affected, and other glands of the cervical region follow, in the severe cases bilaterally. Extension to the axillae may occur. The glands are at first small, insensitive, and arranged in definite groups. Certain ones enlarge, become attached to each other, and to the skin in case suppuration be imminent. After caseation and softening have occurred fluctuation may be detected. Fever is present during the stage of activity, and many of the patients are notably anemic. If not opened, the resulting abscess breaks, leaving an unsightly discharging sinus, and finally a depressed irregular scar.

Although chronic in development in most cases, abrupt onset is not rare and the invasion may be almost universal in distribution. Although the course is usually slow, and tends to recovery, the development of acute miliary tuberculosis or tuberculous meningitis is

not very infrequent. The possibility of such infection after operative measures, though rather remote, must be borne in mind.

**DIAGNOSIS.**—The indolence, chronicity, predominating unilateral infection, tendency to suppuration and sinus and scar formation, absence of decided fever, frequent presence of the so-called scrofulous type of face and frequency of foci elsewhere, suffice for the diagnosis in most instances.

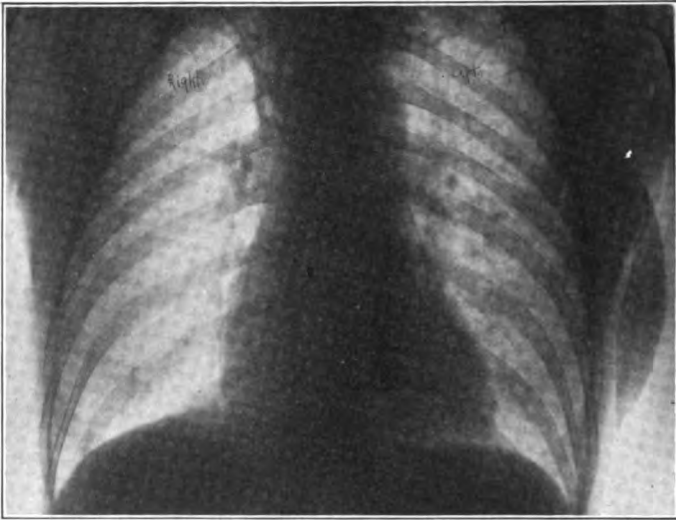


FIG. 24.—OLD HEALED CALCIFIED LYMPHATIC GLANDS. Severe attack of la grippe several years ago, but no clinical suspicion of intrathoracic tuberculosis. (Dr. G. H. Stover.)

**DIFFERENTIAL DIAGNOSIS.**—This involves the distinction from Hodgkin's disease, lymphatic leukemia, simple adenitis and syphilis.

(a) *Hodgkin's Disease.*—The most characteristic feature is the tendency to involvement of other groups of glands, while the tuberculous process is likely to remain stationary in position. The latter affects especially the submaxillary group, and Hodgkin's disease the glands nearer the clavicle. Pseudoleukemia is frequently accompanied by the recurring attacks of fever upon which so much stress has been laid by certain authors. Extensive involvement of the sub-sternal glands points toward Hodgkin's disease, while suppuration

is decidedly indicative of tuberculosis. The tuberculin reaction is presumptively in favor of the scrofulous disease but tuberculosis may co-exist, and indeed Reed and Sternberg find that the tubercle bacillus is present in certain of the cases of Hodgkin's disease, while certain of the patients die of secondary tuberculous meningitis or present gross evidence of tuberculosis at the post mortem. The blood examination gives no certain differential criteria.

(b) *Lymphatic Leukemia*.—This is to be differentiated by the blood examination, the excess of small or large lymphocytes being characteristic. Enlargement of the spleen or liver is frequently present.

(c) *Simple Adenitis*.—This may occur in connection with the infectious diseases of childhood, or diseases of the face, or the ear, the skin, etc. The tendency to subside within a short time is the most valuable diagnostic feature. If suppuration occur it is an acute process rather than a chronic one.

(d) *Syphilis*.—In this disease other glands are enlarged, there are other marks of the disease, the Wassermann reaction is present and treatment is effective in the removal of the adenitis.

**Tracheobronchial Glands.**—The enormous frequency of the tuberculous invasion in these glands in the children of foundling hospitals and in others under unfavorable hygienic conditions is well recognized. It is likely that more cases of tuberculous meningitis originate from this source of infection than from any other. Because of the frequent association of tuberculosis of other glands the exact origin of an acute process is often difficult to determine.

In children the glands may enlarge to such an extent as to press upon the trachea and bronchi, the right primary bronchus, surrounded by the largest group of glands, being especially liable to damage. Perforation of the bronchus may occur. Pressure upon other structures, suppuration, communication of an abscess with a vein, with general systemic infection, and the manifestation of an acute miliary tuberculosis, or extension of the infection to the lung or to the pericardium, are features of the disease in certain cases.

In one of my cases (*Amer. Jour. Med. Science*, Aug., 1899) there was reason for believing that a tuberculous gland was coughed

up after penetrating the left primary bronchus, and many instances are on record of the finding of the gland, loosened by suppuration, as the cause of suffocation, the patient having been unable to force it by cough through the glottis. Traction diverticula of the esophagus occasionally result from the healing and contraction of abscesses resulting from bronchial glands.

**SYMPTOMS.**—These result from the mechanical effects of the glandular enlargement in most cases. Because of the involvement of the recurrent laryngeal and other nerves a chronic spasmodic cough suggesting pertussis is at times noted. Aphonia, and sharp attacks of dyspnea may be present. Cyanosis may originate from the pressure upon the great vessels. I have seen edema of the legs from pressure of tuberculous mediastinal glands upon the inferior cava, and

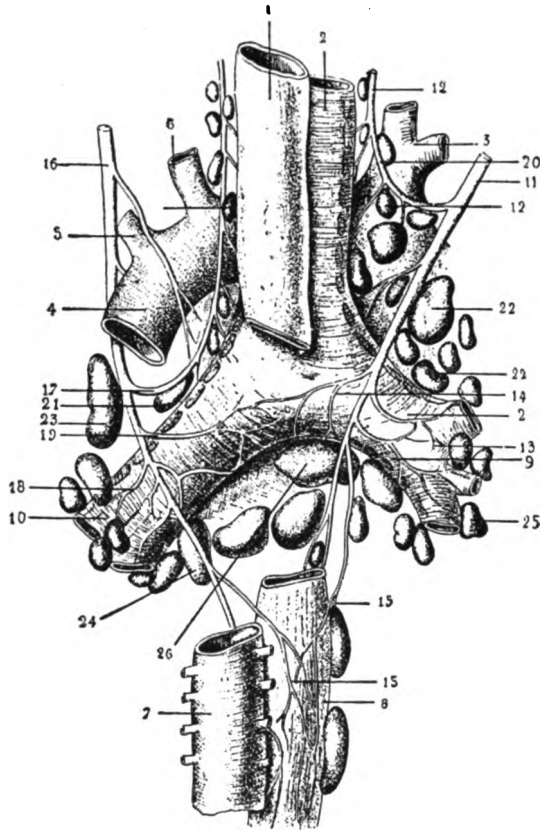


FIG. 25.—THE TRACHEOBRONCHIAL GLANDS. 1. Esophagus; 2. Trachea; 3. Innominate artery; 4. Arch of aorta; 5. Left subclavian artery; 6. Left carotid artery; 7. Thoracic Aorta; 8. Esophagus; 9. Right bronchus; 10. Left bronchus; 11. Right vagus nerve; 12. Right recurrent nerve; 13. Bronchial filaments of right vagus; 14. Anastomotic filaments; 15. Esophageal filaments; 16. Left vagus; 17. Recurrent nerve; 18. Bronchial filaments of left vagus; 19. Anastomotic filaments; 20. Ganglionic chain along the right recurrent nerve; 21. Ganglionic chain along the left recurrent nerve; 22. Right tracheal ganglionic group; 23. Left tracheal ganglionic group; 24. Right inter-bronchial groups; 25. Left inter-bronchial groups; 26. Inter-tracheo-bronchial group. (Charcot's "Traite de Médecine.")



have reported several instances of pressure upon one main bronchus in advanced phthisis, the cornage and tirage of the French authors being present. Dysphagia may occur. It is only in cases of great enlargement or in those where certain glands are favorably situated for production of pressure upon important organs (recurrent laryngeal nerve especially) that characteristic symptoms are produced. I have reported several cases characterized by attacks of hemoptysis extending over many years with cough, fever and many other symptoms of phthisis, but in which no one was ever able to find the physical signs of even the slightest tuberculous infection. It is probable that many such cases may be explained upon the hypothesis of gradually extending bronchial glandular tuberculosis without lung involvement. None of the cases have so far proved fatal to my knowledge, although one has lasted thirty years.

**PHYSICAL SIGNS.**—These are to be detected only in extreme cases, when the enlargement is sufficient to give rise to dulness over the upper sternal region. This is best obtained by light percussion, and is much more reliable than the signs derived from percussion of the upper dorsal region.

In general we may say that the diagnosis of tuberculous infection of the tracheobronchial and mediastinal glands is rather more dependent upon inference and less upon actual demonstration than is the case in most other diseases of the chest.

**DIAGNOSIS.**—This depends upon the signs and symptoms discussed, upon the occasional finding of tubercle bacilli in the sputum, coming either from the associated tuberculous process in the lungs or from the breaking down of a gland with communication with a bronchus; upon a positive tuberculin reaction and the demonstration of the gland by the X-ray. In many of my cases Dr. G. H. Stover has been able to demonstrate the enlarged glands in cases of pulmonary tuberculosis, some of them showing evidence of calcification.

**DIFFERENTIAL DIAGNOSIS.** This involves a discussion of aneurism, sarcoma of mediastinal glands, Hodgkin's disease, leukemia, cancer of the esophagus and substernal thyroid tumor. A careful consideration of the features mentioned above under diagnosis should suffice for the distinction.

**Mesenteric Glands** (*Tabes Mesenterica*).—Enlargement of these glands, with the retroperitoneal adenitis so often associated, is extremely common in children living under conditions of poor nutrition. In many cases the infection probably arises through the intestinal lesions of the digestive diseases of infancy, and in others the involvement is secondary to actual tuberculosis of the intestinal wall. Rapid progress with a fatal result, or caseation and calcification, seem to be more frequent than suppuration. Although pertaining especially to children, this type of tuberculous infection is not rare in adults, and is said to be especially frequent in the colored race, especially in the Northern cities.

**SYMPTOMS.**—Derangement of nutrition, watery diarrhea, hectic fever, irritability, wasting and anemia are present. The abdomen is distended because of the gaseous indigestion, so that in children at least the glands often cannot be detected by palpation. Enlarged and superficial veins are common. An associated tuberculous peritonitis may lead to the development of a palpable tumor, the omentum being then commonly implicated.

**DIAGNOSIS.**—This depends upon the general appearance of the patient, the association of tuberculous lesions elsewhere, occasional detection of the glandular enlargement and the exclusion of other causes (cancer, sarcoma, etc.) of tumor growth in the abdomen. The tuberculin reaction may be decisive, and tubercle bacilli may be found in the stool if open tuberculous lesions of the intestinal mucosa are associated with the adenitis.

#### D. TUBERCULOSIS OF THE SEROUS MEMBRANES

**Pleura.**—The infection of the pleura is generally secondary to tuberculosis elsewhere and may be acute or chronic. In association with tuberculosis of the lungs or tracheobronchial glands an acute dry pleurisy is exceedingly common, oftentimes upon the side of the chest presenting the least involvement of lung. This may be partly explained by the previous occurrence of obliterative pleurisy upon the side of the more advanced process. Next in frequency is the pleurisy with serofibrinous exudate, a variety so common even in sup-

posedly non-tuberculous individuals that a presumption exists in any case with such exudate that it is of tuberculous nature unless another cause is obvious. Hemorrhagic effusion is not rare, and empyema may be found. The presence of pus should suggest careful search for a preëxisting pneumothorax. A fresh fibrinous exudate is present in certain cases, and a chronic fibrous thickening of the pleura may follow in any case.

Acute tuberculous pleurisy may be a part of a general infection of the serous membranes. The serofibrinous effusion is more common early in the course of a pulmonary infection and in many instances the pulmonary process is so entirely latent that it is undiscovered. Many such cases die years afterwards of the lung infection.

The hemorrhagic effusions are of grave prognosis, often passing through a rapidly fatal course.

**Pericardium.**—Involvement occurs in over 4 per cent. of cases coming to autopsy. It is generally secondary, arising from extension from the pleura or occasionally from perforation of the pericardium by a tuberculous cavity. The varieties correspond with those noted in inflammation of the pleura. The diagnosis depends upon the recognition of the tuberculous nature of the primary process and the chronic course.

**Peritoneum.**—This membrane may be involved in the acute tuberculous infection of the serous membranes. Over tuberculous ulcerations of the bowel, circumscribed areas may be found in which the serous coat is thickly dotted with miliary tubercles. In the appendix and cecum the location of the ulcerations within may often be thus defined.

In the acute infection a serofibrinous or bloody exudate may be present. In the chronic forms, great thickening of the peritoneum and omentum are common,—a chronic proliferative peritonitis. The frequency of infection from the fallopian tubes should be recognized. Tuberculous peritonitis is a common infection in cirrhosis of the liver. Tuberculosis of the lungs exists in a majority of cases of peritoneal infection. The subject will be further discussed under diseases of the peritoneum.

## E. TUBERCULOSIS OF THE DIGESTIVE TRACT

Tuberculosis of the lips, gums, tongue, salivary glands and hard and soft palate and pharynx is not very infrequent, especially in association with tuberculosis of the upper air passages. The chronicity of the ulceration upon the lips and tongue leads to a mistaken diagnosis of syphilis in certain cases. Absence of adenitis at the angle of the jaw is in favor of the tuberculous infection.

In addition to the superficial ulceration, deep tuberculous infiltration may occur. In the tonsillar infection marked cervical adenitis is present, and the "scrofulous" glands of children are probably most often secondary to such infection. Histological examination of the tissue or the finding of the bacilli generally suffices for the diagnosis. In case of doubt as to syphilis the laboratory procedures must be invoked. The course is likely to be extremely prolonged. I have known a tuberculous ulceration upon the frenum of the tongue to persist without much change for more than five years.

The esophagus and stomach are not often affected. The dysphagia associated with laryngeal tuberculosis is rather from the pharyngeal involvement often present than from affection of the esophagus. Blumer believes that the acidity of the gastric secretions preserves the stomach from infection in most cases. Pyloric ulceration is most common. It may be almost devoid of symptoms. Acute miliary infection may occur.

**Intestinal Tuberculosis.**—Brown states that this is the most frequent complication of chronic pulmonary tuberculosis. It is so often latent that the frequency is much greater if judged by post mortem statistics than would be indicated by clinical records.

Intestinal tuberculosis may be primary in children, being the initial step in *tabes mesenterica*.

**SYMPTOMS.**—These are uncertain and there may be none recognizable. Diarrhea is common if the ulcerations are low in the digestive tract, but it is frequently absent in other cases. Constipation may exist; hemorrhage rarely occurs, but death may result from it. Irregular fever is suggestive of tuberculosis in case of doubt.

The secondary tuberculous infection of the intestine is generally

seen in the large bowel or lower part of the small one, and commonly comes from the swallowed sputum. The more or less annular ulcerations are characteristic. The mesenteric glands are often extensively involved. Colicky pains and diarrhea are common features. Perforation of the bowel may take place, or complete or partial obstruction, either from cicatricial contraction at the site of a large ulcer, or from partial distortion with such gluing together of adjacent loops of bowel as to prevent the passage of feces, in part mechanically and in part dynamically. I have seen complete and lasting recovery after operation in such a case, but less complete recovery with death in a few months in several others. Four large ulcers of the ileum were found accidentally at operation in the case of an old lady with intestinal obstruction from old appendiceal adhesions, but no definite symptoms from their presence were noted before or after operation.

Great interest attaches to the development of tuberculous lesions in the region of the appendix and cecum. A hypertrophic form, with great thickening, connective tissue hypertrophy, and resulting narrowing of the lumen, is occasionally mistaken for a malignant growth. It is said to be more common in males of middle age with slowly advancing tuberculosis. Perforation externally with resulting fistula may take place.

Non-tuberculous appendicitis occurring in tuberculous patients is spoken of in the Chapter upon Appendicitis. The tuberculous variety is fairly common, ulcers being found within the appendix with miliary tubercles upon the overlying serous coat. I have noted several such cases of fairly acute onset, so that the real condition was not suspected until operation. Primary tuberculous appendicitis has been reported.

It is more common to find the affected appendix involved in a great mass with the tuberculous cecum, with infected glands adjacent. Favorable cases of this type have proved to be fair operative risks, there being 48 deaths in Hartman's 229 cases reported from the literature.

Abdominal pain and tenderness, tumor formation, obstructive symptoms, distention of partially obstructed loops of bowel, especially of the colon, local "doughy" feeling upon palpation, and per-

haps a general tuberculous peritonitis, are features of advanced intestinal tuberculosis. Fistulous openings may develop. The patent urachus may become infected.

**Fistula in Ano.**—This occurs in about 3 per cent. of cases of pulmonary tuberculosis. It begins as an ischiorectal abscess, and may first call attention to the existence of tuberculosis. The failure to heal after operation is very suggestive of the infection, even though no other signs of tuberculosis have been noted. If opened early, perforation into the rectum may be avoided in some cases. A fistula is not necessarily tuberculous in the consumptive. Pain upon defecation, upon sitting upon some surface which permits of pressure upon the part, or upon coughing, is a prominent symptom.

**DIAGNOSIS.**—The finding of the tubercle bacilli in the feces does not at all prove the presence of intestinal tuberculosis. If it can be proved that the disease of the bowel is primary, the finding of the bacilli is positive evidence, but this cannot often be the case. Tubercle bacilli in the discharges from a fecal fistula are very suggestive. In general the diagnosis must rest upon the features outlined in the description given above. The differentiation of intestinal tuberculosis from non-tuberculous appendicitis has been considered. Cancer of the bowel presents many points of similarity. Pain is commonly a more prominent feature and fever is less conspicuous. Stenosis is more common in cancer. If bacilli occur in the stools in the absence of a pulmonary or other source, the diagnosis is practically certain. The tuberculin reaction would be of some value in such a case.

#### F. TUBERCULOSIS OF OTHER ORGANS

**Liver.**—Secondary infection of the liver is fairly common, the vascular channels favoring it. Tuberculous lesions along the course of the mesenteric veins are especially likely to be followed by the hepatic infection, and the tubercles may be found widely disseminated in its structure. The peritoneal covering is often involved in peritoneal tuberculosis.

**SYMPTOMS.**—These are not distinctive. Enlargement of the liver may occur, but in the absence of palpable tuberculous masses is not

to be differentiated from the fatty infiltration or amyloid enlargement often found in advanced tuberculosis. Pain may occur if the peritoneum be involved.

**Spleen.**—Although frequently involved, tuberculosis of the spleen is commonly incapable of clinical diagnosis. Polycythemia has been present in a few cases.

**Brain.**—Tuberculosis of the brain and cord will be considered in the section upon the nervous system.

**Tuberculous Arthritis.**—For a consideration of the infections of the joints the reader is referred to the works upon surgery.

**Tuberculous Laryngitis.**—This occurs as a secondary process in approximately 30 per cent. of the cases of pulmonary tuberculosis. Tuberculous infiltration occurs in the epiglottis and the arytenoid region most frequently. It is followed by ulceration and secondary infection and often by involvement of the cartilaginous structures. Cough, hoarseness, aphonia, marked and troublesome dysphagia, tenderness over the larynx and local adenitis are present. Syphilis and cancer must be considered in the differential diagnosis. For a fuller consideration the reader is referred to the special works.

#### G. TUBERCULOSIS OF THE GENITO-URINARY ORGANS

The testes in the male and the fallopian tubes in the female are most frequently involved. The testicular involvement may be unilateral or bilateral, and is commonly secondary to the lung or other infection, being often associated with peritoneal tuberculosis. The epididymis is especially affected. It is rarely acute in onset, but may be, as in one of my cases, so abrupt in development as to simulate the gonorrheal form of infection. In these cases hydrocele may be noted, and extension to the opposite side is to be feared. The chronic form is associated with swelling, especially affecting the epididymis, and with less severe pain and tenderness than are present in the acute type, and with extensive caseation, often followed by external perforation and the formation of a persisting tuberculous fistula. I have known caseation to be followed by such calcification that the whole testis many years afterward was literally of stony hardness.

The tubes are very commonly involved, often primarily, and are the most frequent origin of a secondary tuberculous peritonitis. The ovary may also be infected secondarily. Bilateral salpingitis is often present. The uterus is rarely implicated.

The prostate and seminal vesicles are not infrequently infected. The organs become enlarged, nodular and tender. The very frequent association of the infection in these organs with that in the bladder and kidneys should be noted. The infection may be of ascending or descending type.

The bladder is commonly infected secondarily, from some other lesion in the genito-urinary tract, generally from one kidney. A tuberculous process in a neighboring organ may invade the bladder by direct extension through its wall. The small nodules enlarge, become caseous, break down and leave ulcers on the mucous surface, these often enlarging by coalescence with neighboring ulcers. Vesical irritability, pain, the presence of pus and often of blood in the urine, with possibly the recognition of tubercle bacilli in the sediment, with absence of signs of stone, are the diagnostic criteria. Many patients unfortunately are treated for cystitis for months when the true diagnosis is an irritable bladder associated with tuberculosis of the kidney. The loss of time is a serious matter because of the opportunity for development of tuberculosis in the bladder.

**Suprarenal Capsules.**—Tuberculosis of the adrenals, especially of the fibrocaceous variety, is the most common cause of Addison's disease. The diagnosis is possible only as it is based upon the symptoms of that affection. In one case, one kidney was removed for tuberculosis, and after a period of health lasting six years Addison's disease developed and we found the characteristic tuberculous lesions of the remaining capsule at the post mortem examination.

**Kidneys.**—Apart from acute and chronic nephritis, amyloid disease and bacillary infection without evidence of tuberculosis, all of which may complicate pulmonary tuberculosis, renal tuberculosis is often found, probably in 5 per cent. to 10 per cent. of all cases. Pathologically speaking, the disease is bilateral in 60 per cent. of the cases coming to autopsy, but clinically it is unilateral in much more than half the cases reported. Primary renal tuberculosis is



not uncommon, although the infection of other portions of the genito-urinary tract is so commonly associated that it is difficult to be certain of the original seat of the trouble. Males are most often affected. Miliary tubercles are numerous in the cortex in acute miliary tuberculosis, but death occurs in this form before diagnosis is possible so far as the renal complication is concerned.

In the chronic form of renal tuberculosis the tubercles appear in the substance of the kidney, break down and coalesce so as to form an abscess or multiple abscesses. Commonly the abscess breaks through into the pelvis of the kidney, and a tuberculous abscess may surround the kidney in this case, or extend to other organs. Caseation and calcification may occur.

**SYMPTOMS.**—Increased frequency of urination, and passage of pale, abundant urine, containing albumin, pus and epithelial cells, are prominent features. Irritable bladder was noted by Braasch in 86 per cent. of his cases. Tube casts may be present. The urine may be cloudy from pus, or somewhat smoky from the presence of blood, which is found in 60 per cent. of the cases. The subsidence of the sediment in a flat-topped layer, with sharp margin, is often to be noted. The tubercle bacilli may be isolated with care in a majority of the cases. The smegma bacillus must be carefully differentiated. Mixed infection, often with the colon bacillus, is common. Failure to demonstrate the tubercle bacilli should not be regarded as of great weight, since they are often temporarily absent.

Pain is often wanting. It may be more prominent upon the unaffected side. Paroxysms resembling those of mild renal colic attend the passage of clots, necrotic tissue or debris through the uréter. The kidney is often tender upon palpation, and may be felt to be enlarged (20 per cent.—Braasch). A moderate blow with the hand over the lower ribs covering the affected kidney is often distinctly painful. The pain in the bladder may arise from the irritation of that viscus, or because of the secondary tuberculous infection.

Chills, fever, sweating, loss of flesh, failure in general condition, and irritability, often in part accounted for by the broken rest because of frequent micturition, soon follow.

**DIAGNOSIS.**—In the presence of the symptoms and signs men-

tioned, the finding of other foci of tuberculosis may be looked upon as justifying the diagnosis of renal tuberculosis. In the absence of such corroborative evidence, a sharply positive tuberculin reaction will commonly suffice. Cystoscopic examination adds greatly to the probability of the diagnosis if it demonstrates a reddened, swollen ureteral opening upon the suspected side. The presence of presumably tuberculous ulcerations in the bladder would also be valuable as evidence.

Ureteral catheterization may demonstrate not only which kidney is affected, but whether the other one is sufficiently active to justify operation. The question of danger of carrying infection by the use of the ureteral catheter must be submitted to the operator.

Inoculation experiments performed upon guinea pigs with the sediment obtained from the urine drawn by the ureteral catheter might demonstrate that tuberculosis existed, and which kidney or ureter was involved, but the use of sediment from urine drawn from the bladder by the catheter would not prove the exact location in the urinary tract of the tuberculous lesion. On the other hand, we must note that tubercle bacilli have been found in the urine in pulmonary tuberculosis without any gross lesion being demonstrable in the urinary tract at the autopsy, having evidently passed through the glomeruli of the kidney with the secretion.

The Röntgen-ray examination often demonstrates the existence of a shadow, commonly in one pole of the kidney, generally larger and much less sharp in outline than is caused by a stone. This shadow of the infected area cannot be distinguished from that of a renal tumor, but may be of much value, nevertheless, in pointing out with certainty the affected kidney.

The presence of irritative and degenerative changes in the supposedly unaffected kidney, as shown by the examination of the segregated urine, does not at all preclude a consideration of the wisdom of operative measures upon the tuberculous one.

**DIFFERENTIAL DIAGNOSIS.**—This must take into consideration calculous pyelitis, renal neoplasm and intermittent hydronephrosis. The first is commonly attended by greater pain, especially in colicky attacks, by greater liability to hemorrhage, and by greater tender-

ness of the kidney. Crystalline material and epithelial cells are commonly present in the sediment and the X-ray may show the stone.

In the hemorrhagic cases only is renal neoplasm, especially hypernephroma, to be seriously considered. Fever, colicky pain, and pyuria are often wanting, and the X-ray may demonstrate the tumor.

An intermittent hydronephrosis with mixed secondary infection may closely simulate renal tuberculosis, as in a case of mine just operated upon. Colicky pain, sharp hemorrhage, enlargement of the kidney, tenderness, fever, frequent urination, purulent urine laden with bacteria, with blood at times, and the loss of weight and general condition so prominent in renal tuberculosis were all present. In the absence of positive evidence of any kind that tuberculosis was present the correct diagnosis was made before the operation.

**PROGNOSIS.**—This is grave excepting in those cases in which the disease is practically confined to one kidney, which can be safely removed. The downward course in other cases is slow and progressive. About 30 per cent. die of tuberculous meningitis.

### III. LOBAR PNEUMONIA

**Definition.**—Lung fever is an acute infectious disease characterized by fever and inflammation of the lung, and caused, in a majority of cases, by the *Diplococcus pneumoniae*. It occurs everywhere but most frequently in the temperate zone. It is one of the most important diseases, since it represents 2 per cent. to 4 per cent. of all internal disease, and causes 9 per cent. or 10 per cent. of all deaths. It is more common in cold weather, especially in cold changeable weather. It is often seen in epidemic outbreaks, several members of one family, or many of the inmates of the institutions, being affected. I have treated at the same time three members of one family with pneumonia on two different occasions. The virulence seems to increase as the epidemic progresses in many instances. Its transmissibility is well established. Even the inhalation of dust from sweeping public buildings, as by janitors, carries a definite danger of infection, as in the case of tuberculosis. The effect of

cold has been demonstrated by laboratory experiments upon animals infected by the pneumococcus and then subjected to chilling.

Alcoholism definitely predisposes to acute pneumonia. Acclimation offers a certain degree of protection against it. Chest injuries are frequently followed by the disease. No immunity is established. On the contrary, repeated attacks are not infrequent. I took care of one patient through her fourth, fifth, sixth and seventh attacks.

**Bacteriology.**—The *Diplococcus pneumoniae* is found in probably 75 per cent. of the cases. It may be active in other cases in which Friedländer's bacillus, the streptococcus, the staphylococcus, etc., are found most abundantly. An inflammation of the lung indistinguishable clinically from that caused by the pneumococcus may be caused by either of these organisms, or by the typhoid bacillus, influenza bacillus, plague bacillus, or diphtheria bacillus, although pathologically the lung condition is found to be more frequently of the bronchopneumonic type if organisms other than the *Diplococcus pneumoniae* be the cause. Nevertheless, the clinician must class the cases presenting lobar involvement with the usual signs as lobar pneumonia, although the laboratory may not establish the *Diplococcus pneumoniae* as the cause. The failure of the various sera introduced for the treatment of pneumonia is at least in part attributable to failure as to exact bacteriological diagnosis, although perhaps also because of the greatly varying virulence of different strains of pneumococci.

Because of their influence upon diagnosis it is necessary to speak briefly of the pathological changes in the lung in lobar pneumonia. The stage of engorgement, in which the lung tissue is filled with blood, all the capillaries being distended, with perhaps some fibrinous exudate in the smaller bronchi, lasts from 12 to 24 hours, and is recognized clinically by the fever and other general symptoms, and upon physical examination by the dulness, suppressed respiration and perhaps crepitant rales. The second stage, that of red hepatization, is characterized by the distention of the alveoli and smaller bronchi with the croupous exudate, and is the stage in which the characteristic physical signs of acute pneumonia are found. (*See below*). The third stage, that of beginning resolution (gray hepatic-

zation) shows the lung of a dirty gray color, with beginning and later progressing softening of the exudate, abundant leukocytes displacing the red cells in the alveoli. Purulent infiltration of lung, abscess or gangrene, or fibroid induration may complicate resolution or appear as sequelae.

**Symptoms.**—The stage of incubation is brief, but the exact time is not established, and is doubtless quite variable. It is not uncommon to see those who have nursed cases of pneumonia attacked within three or four days. Several examples have fallen under my observation.

In general the more nearly an attack of pneumonia follows the course to be described the more confidently we may ascribe it to the pneumococcus. The onset is abrupt, with severe and prolonged chill, high fever, cough, pain in the side, headache and malaise, delirium, increased respiratory rate, expiratory grunt, and rusty-colored sputum. I have seen all these features present in less than three hours from the first intimation of the illness, though this is very unusual.

**TEMPERATURE.**—The temperature commonly reaches 104° F. to 105° F. within a few hours, and remains high, with nocturnal remissions of a degree or two, until the crisis. In atypical cases, in alcoholics and the debilitated, the fever is often much less pronounced, and in the senile form of the disease, may require careful and repeated observations for its detection, or it may be absent. I have seen it absent throughout in a strong middle-aged adult with all of the usual physical signs and abundant pneumococci in the sputum. It is often less in children than in adults. A pseudocrisis may occur during the course of the fever. In typical cases the temperature falls by crisis at some time between the third and the thirteenth to fifteenth day, the fall being more likely to occur on the seventh, eighth or ninth day. The crisis is accompanied by sweating, and is likely to occur during the night, the temperature falling to normal or below in three to twelve hours. I have seen a continuous fall of ten degrees with recovery. Prolonged sleep may accompany or follow the crisis, and is of good significance. Collapse may supervene upon the critical fall, so that the temperature never rises again from the low point in case of fatal result. Absence of typical

febrile course in pneumonia adds nothing to the patient's chances of recovery.

**COUGH AND EXPECTORATION.**—Cough is likely to be very painful, frequent, short, and in the early stages dry. It may be absent in the asthenic cases and in others in which pleural involvement is absent. After the first day or two in typical cases, an extremely viscid, red, or streaked sputum is raised, often with much difficulty and not exceeding two to four ounces daily. Free hemoptysis occurred in 4 out of 250 reported private cases.\* It is probably more frequent than is commonly believed. After resolution begins, expectoration becomes more abundant and more purulent. Persistent cough after the crisis should lead to examination for pleural effusion. A "prune juice" expectoration is of bad significance. The sputum may be yellowish if jaundice be present.

**PAIN.**—This is of pleuritic origin, chiefly if not entirely, and is sharp, stitch-like, and referred, more or less regardless of the exact site of the inflammation, to the region of the nipple in a majority of the cases. If the lower lobe be involved, and more especially in children, a diaphragmatic pleuritic involvement is frequently present and the pain is reflected into the abdomen, almost always upon the affected side. I have reported an instance, however, of pain in the right abdomen leading to a preliminary diagnosis of appendicitis, from a left-sided pneumonia. Rigidity of abdominal muscles commonly accompanies the pain.

The pain is most severe during the first two or three days of the attack, is aggravated by cough and deep respiration, is commonly lessened by lateral decubitus upon the affected side, which lessens respiratory movement, is less in apical pneumonia, and often absent in senile and exhaustive types of the disease.

**DYSPNEA.**—The respiratory rate is doubled or trebled, and much distress may be caused by the inability to obtain sufficient air. The ratio between respiration rate and pulse rate may rise to one to two or even higher. The increase in the rate because of pleural pain often decreases markedly upon the administration of an anodyne. That the increased rate is not due primarily to the mechanical ob-

\*Report of Cases of Pneumonia in Colorado, Trans. Amer. Climatol. Soc., 1909.

struction in the lung is proven by the fall to normal at the time of crisis, though an entire lung may still be solid.

**CARDIAC PHENOMENA.**—The pulse commonly rises to 100 or more, a rise to 130 or 140 being of grave significance. The full bounding pulse of the typical acute pneumonia during the first days is perhaps as characteristic as the dichrotic pulse of the first week of typhoid. At the time of the initial chill the pulse may be small and may again become small and feeble as consolidation progresses. Irregularity is not uncommon, and is of very unfavorable omen. The arterial tension is slightly decreased in the average cases, but the pulmonic tension, as shown by the sharp accent of the pulmonary second sound, is often raised. A failure of this accentuation with increased frequency of the pulse and cardiac dilatation signifies danger. The heart dilates in severe cases and relative leakage at the mitral orifice is frequent. Severe toxemia, prolonged high fever, and extensive involvement, especially if upon the left side, are likely to be followed by cardiac failure.

**BLOOD.**—A polynucleosis is found in most cases, reaching 10,000 to 30,000 in average cases, and often even 40,000 to 50,000. A failure of disappearance at crisis or soon after should lead to search for a complication. The fibrin content of the blood is increased. A failure of the leukocytosis in a typical case is cause for great anxiety.

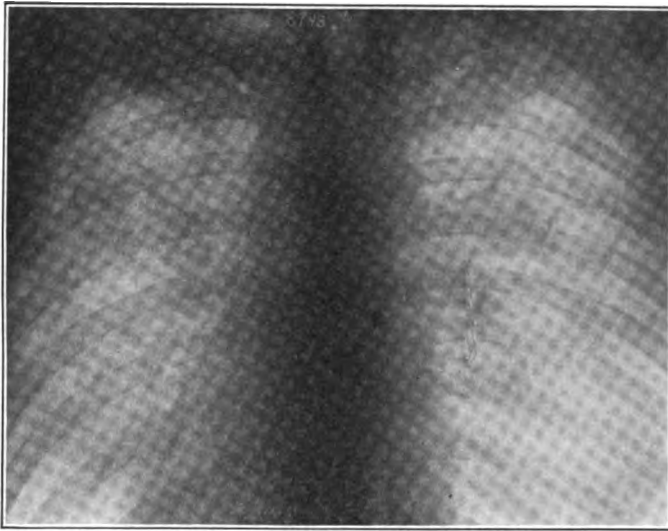
**NERVOUS PHENOMENA.**—Convulsions frequently take the place of the chill in children. Delirium occurs in ten to twenty per cent. of the cases, and with vastly greater frequency in the fatal ones than in those recovering. It may vary from the muttering type in feeble patients to the violent maniacal form in sthenic cases. Delirium tremens occurred in 4.26 per cent. of 8,998 collected cases with a fatality of nearly 37 per cent.

Insomnia is not infrequent. Apical pneumonia is distinctly more liable to nervous complications. The meningeal symptoms often noted in children do not at all signify an actual meningitis in most instances. (Cerebral pneumonia.)

**DIGESTIVE SYSTEM.**—Vomiting is not infrequent at the time of the onset. Complete anorexia is the rule for the first few days. The tongue is commonly white and deeply furred, later becoming red and

glazed, or, in the severe cases, brown, dry, and cracked. The abdomen is often distended with gas, adding to the respiratory distress by crowding up the diaphragm. Constipation is the rule. The spleen is frequently moderately enlarged. Hiccough is occasionally very distressing.

**SKIN.**—Herpes labialis is found in probably one-fourth to one-half the cases and is of definite diagnostic value in distinguishing between pneumonia and typhoid, being rare in the latter disease.



**Fig. 26.—FOCAL PNEUMONIA.** Röntgenogram made just before attack of croupous pneumonia. (Dr. G. H. Stover.)

Abundant sudaminal eruption is not infrequent. A localized redness of the cheek, often accompanied with contraction of the pupil, is frequently noted on the side of the involvement. Cyanosis of slight degree is common in the earlier stages and is constantly found in severe cases going on to a fatal ending. The color may be almost that of a concord grape in the worst cases. Sweating is profuse at the time of crisis. Petechiæ may be noted in septic cases. Slight jaundice is not infrequent.

**URINE.**—The secretion is scanty, of high color and specific gravity, and contains practically no chlorids during the exudative stage.



Febrile albuminuria is present in probably half the cases. Acute nephritis may develop.

**Physical Examination.**—The diagnosis of acute pneumonia may practically be made by inspection in many cases, so characteristic are the findings. The flushed face, often with contracted pupil, the frequent herpes labialis, bounding arteries, obvious dyspnea, frequent cyanosis, free movement of the *alæ nasi*, lateral decubitus or propped-up position in many severe cases, lack of movement of the affected side, with the expiratory grunt and the appearance of the sputum cup, suffice for a presumptive diagnosis.

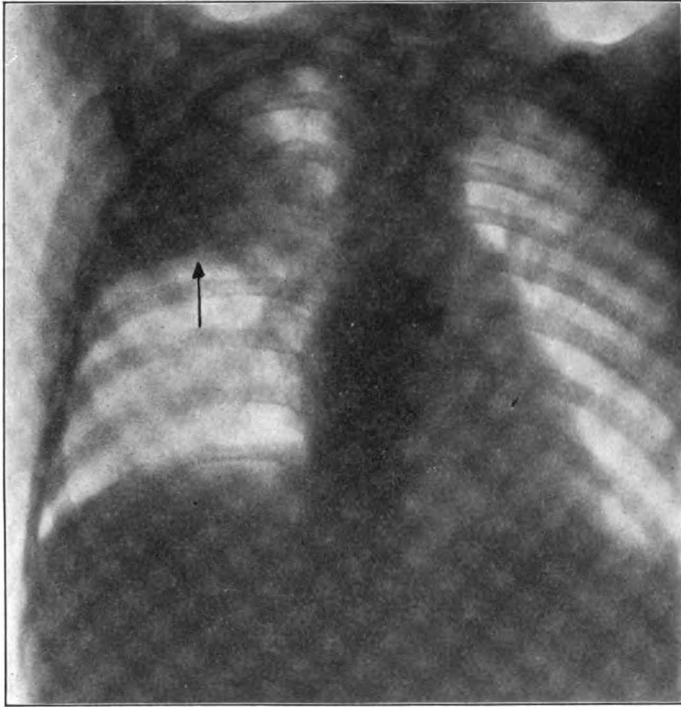
**PALPATION.**—This may show decreased respiratory movement of the affected side. Tactile fremitus may be increased over the area involved, if the bronchi be clear, or absent if they be filled with exudate. (Massive pneumonia.)

**PERCUSSION.**—Skodaic resonance is often found early, and especially in the upper part of the chest. If the patient be recumbent, with the pneumonic process limited to the posterior portions of the lung, the skodaic resonance may be almost startling in intensity upon percussing the anterior portions. Cracked-pot resonance is not infrequent in children over the apices. The characteristic finding is that of dulness over the area involved, which reaches absolute flatness in massive pneumonia. The dulness may have a definite lobar distribution. A gradually increasing dulness may be detected in many cases of central pneumonia as the focus of consolidation approaches the surface.

**AUSCULTATION.**—A pleuritic friction sound is often heard. The respiratory sounds are partially suppressed in the congestive stage, and may be wholly absent in massive pneumonia. Bronchovesicular and finally intensely bronchial respiration may follow. The crepitant rales appear at the end of inspiration as the exudative stage begins, and reappear (*crepitus redux*) as resolution begins. During the height of the croupous exudative stage the respiration is typically tubular and dry, with marked bronchophony. Subcrepitant rales, from the presence of exudate in the smaller tubules, may be present at practically any stage. The bronchial breath sounds may be transmitted to some extent to the sound side. Moderate dulness and a

few moist rales are frequently heard over the base of the sound lung, as a result of the collateral congestion, and without implying pneumonic involvement.

As resolution progresses abundant coarse moist rales are commonly present. The dulness gradually decreases as more air gradually enters the alveoli, bronchovesicular respiration replaces that of the



**FIG. 27.—CENTRAL PNEUMONIA OF THE RIGHT UPPER LOBE.**(Indicated by arrow.) Dark spot over spine is shadow of a marker. Repeated physical examinations by Drs. Kleiner and Hall, both before and after the picture was taken, showed practically no physical signs. Patient aged 9 years. (Dr. S. B. Childs.)

bronchial type, and gradually gives way as convalescence advances to normal respiration.

**Clinical Varieties of Pneumonia.**—(a) The ordinary type has been described.

(b) **ABORTIVE PNEUMONIA (Larval Pneumonia).**—This occurs as a typical but extremely short attack, especially in children. Full

consolidation of the affected lung does not take place. Marked dullness and high fever may be noted in a few hours after the onset. In 24 to 72 hours the fever subsides, often by crisis, and convalescence begins. The possibility of the presence of this type of pneumonia should be considered in estimating the value of any form of treatment.

(c) **LATENT PNEUMONIA.**—The usual manifestations of the disease are occasionally almost absent, especially in the aged and debilitated. In most instances the disease is probably central in the lung, accounting, especially in the presence of emphysema, for the absence of physical signs and of pain. The respiratory and pulse rates are but little raised because of the mildness of the attack. The rule should be absolute to examine the chest with especial care in the aged and feeble, when great weakness or slight cough, dyspnea, cyanosis or delirium are noted. Accentuation of the pulmonary second sound and the finding of the pneumococcus in the sputum are often of value in the diagnosis.

(d) **TERMINAL PNEUMONIA.**—Many old and feeble patients with various chronic diseases die with a terminal pneumococcic infection. Probably half of those passing seventy years die in this manner. The type is likely to be that just described as latent.

(e) **INTERCURRENT PNEUMONIA.**—This adds the features of pneumonia, generally moderate in severity, to those of the disease present. It is most common in typhoid, and frequently affects the lower lobes because of the already existing passive congestion there. I have seen four lobes involved in succession at intervals of several days in typhoid, with eventual recovery. This type of pneumonia is the cause of the great mortality in epidemics of influenza. In diphtheria, typhus, small-pox, and other acute infectious diseases it is a common co-infection.

(f) **SENILE PNEUMONIA.**—This is likely to be of the latent type mentioned and is often practically without symptoms which would attract attention unless the facts that the aged so commonly die of pneumonia and that the usual manifestations are absent be borne in mind.

(g) **TYPHOID PNEUMONIA.**—This is an unfortunately named

variety, distinguished by its low typhoidlike course, and found in those with chronic disease, in septic conditions, etc. It should be clearly understood that the *B. typhosus* plays no part in its origin. Diarrhea often occurs in this type of general infection by the pneumococcus, or by this organism in association with the streptococcus or other secondary invaders, and overshadows the symptoms dependent upon the local pneumonic process. It is commonly of gradual or even concealed onset, without the symptoms typical of uncomplicated lobar pneumonia, and with more stupor, tremor, and delirium than characterize the latter disease. Prostration is marked and the prognosis is grave.

(h) **APICAL PNEUMONIA.**—This implies apical localization of the inflammatory process and is often complicated with delirium and high fever, and with hiccough. It occurs more frequently in children. The prognosis does not seem proven to be especially worse than in other types.

(i) **DOUBLE PNEUMONIA.**—This is not infrequent, the extension generally occurring during the first one or two days of the disease. The increased local involvement, the commonly longer course, and the greater strain upon the right heart add to the gravity of the attack.

(j) **MIGRATORY PNEUMONIA.**—This term relates to the movement of the pneumonic focus from one part of the lung to another or to the opposite lung. There is a failure of crisis in many such cases, and the outlook is grave. The “creeping” is unattended with chill or other phenomena of new infection. The patient fails to form the usual antibodies which put an end to the extension.

(k) **MASSIVE PNEUMONIA.**—This term signifies the type in which the bronchi are solidly filled with exudate, so that no air enters the part involved. The physical signs are those of an effusion, excepting that no displacement of surrounding organs occurs.

Certain terms often used should be defined. By endemic pneumonia we mean the type in which several members of the same household have the disease at once or in succession. By epidemic pneumonia we understand the extensive outbreaks occurring in towns, camps, institutions, etc., in which perhaps a fourth to a half

of the population is attacked. Some especial variety of pneumococcus may characterize the epidemic. Postoperative pneumonia is of frequent occurrence especially after serious operations, and more especially about the mouth. Many patients operated upon for abdominal diseases, and especially for cancer in the upper abdomen die of pneumonia. A separate type is anesthesia pneumonia. This is more common after ether than other anesthetics, and dependent upon the chilling and irritating effects of the vapor inhaled, upon the lowering of the body temperature and of the resistance, in a feeble individual, from the anesthetic, and in part, perhaps a predominating part, from inhalation of the fluids from the mouth and nose, of food in postanesthetic vomiting, etc. It may thus be a true inhalation pneumonia such as we have seen in those who have inhaled fluids into the lung, or swallowed food, etc., after loss of control of the larynx and pharynx, in the insane and others. Most of these pneumonias are rather of lobular than croupous type. In the true lobar form the pneumococcus may often be demonstrated in the fluids from the mouth, and it has presumably started into infective activity under the favoring circumstances of the anesthetic and the operation. Anders found that 80 per cent. of ether pneumonias occurred in cold weather. About one-third of one per cent. of cases operated upon under anesthesia, in large groups of cases he studied, developed pneumonia.

Relapsing pneumonia signifies, of course, that the disease relapses, though this is very rare, and the term is scarcely called for. The term influenzal pneumonia relates to the occurrence of pneumonia in association with grippe.

**Complications.**—**DELAYED RESOLUTION.**—This is much less frequent than formerly stated, as many of the supposed cases are due to small purulent pleural effusions, especially between the lobes, or other complications. The term should probably be confined to those frank cases in which the fever subsides but the physical signs of the disease remain, for two or three weeks, eventually resolving. The type in which the signs remain, with a more or less septic form of fever, and occurring after typical forms of pneumonia, must be often due to small foci of suppuration in lung or pleura, and should be

regarded rather as a septic complication of the disease. A fibroid interstitial pneumonia may develop after the acute disease has disappeared. Phthisis florida is an occasional sequence.

Pleural effusion occurs in about 6 per cent. of the cases.\* Since pneumonia is commonly a pleuropneumonia, this is not surprising.

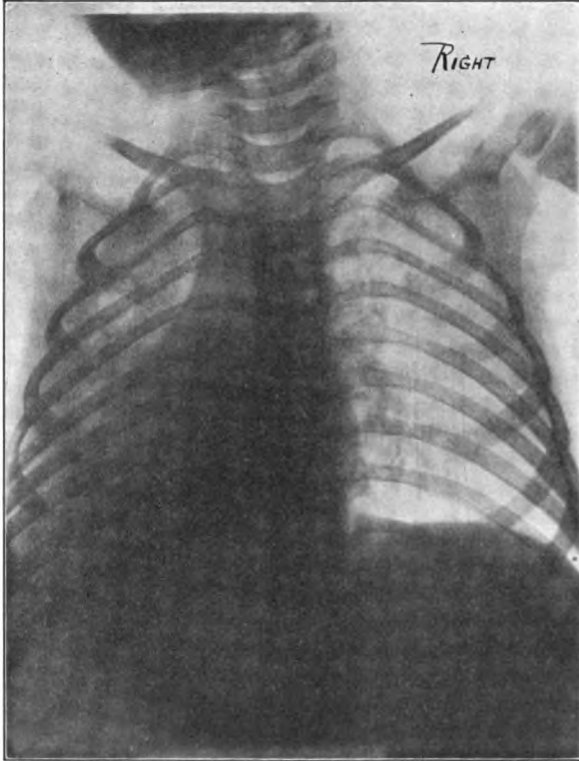


FIG. 28.—POSTERO-ANTERIOR VIEW OF THE CHEST OF A CHILD, SHOWING A PNEUMONIA OF THE LEFT LUNG IN THE PROCESS OF RESOLUTION, FOLLOWING THE REMOVAL OF A BEAN FROM THE LEFT COMMON BRONCHUS. Patient made a good recovery. (Dr. S. B. Childs.)

In rather over 2 per cent. empyema, most often pneumococcic, but often streptococcic, is found as a sequel. I have noted it with especial frequency in those who have suffered from a painful diaphragmatic pleurisy. Pericarditis is not rare, and is especially fre-

\*Osler: "Modern Medicine."

quent in left-sided pneumonia. It is found at the post mortem examination with much greater frequency than in the sick room. Amongst other complications we should note simple endocarditis, malignant endocarditis, acute nephritis, abscess of lung, gangrene of lung, meningitis, parotitis, arthritis, hepatitis, jaundice, especially in involvement of the right lower lobe, otitis media, peritonitis and pulmonary thrombosis. Multiple neuritis may occur as a sequel. In the series of personal cases quoted there were as complications two cases of post-febrile bradycardia, two of cerebral thrombosis, one of pneumothorax, one of pulmonary thrombosis, and one of post-febrile melancholia. Subphrenic abscess, mediastinitis and enlargement of the bronchial glands are rare complications. Almost any septic complication is possible in pneumonia.

**Diagnosis.**—In frank cases this is rarely in doubt. In the senile, septic and other atypical forms the diagnosis fails rather from lack of careful physical examination than otherwise. We should repeat that pneumonia is so common in protracted illness in debilitated subjects that frequent examination should be made, since it is almost sure to pass unrecognized if we await symptoms calling attention to its presence.

The young physician is occasionally deterred from making the diagnosis by the lack of physical signs. These may be wanting since a focus of consolidation of the size of an egg in the center of the lower lobe, for example, cannot be found, and yet it is abundantly able to give rise to all the symptoms. Given a history of chill, fever, pain in the chest, cough, with leukocytosis, herpes labialis and red expectoration, physical signs are not necessary to the diagnosis. In many cases the laboratory examination is practically decisive. The value of blood cultures must not be overlooked.

**Differential Diagnosis.**—(a) ACUTE PNEUMONIC PHTHISIS.—Since this may occur suddenly in apparent health it is easily and generally mistaken for acute pneumonia during the early days of the disease.

The history of cough and loss of weight, or of marked family predisposition may awaken suspicion. More marked remissions in temperature, the occurrence of chills and sweats and the failure of

crisis at the expected time should lead to an examination of the sputum for tubercle bacilli. They have been found as early as the middle of the first week. Elastic tissue appears shortly, the expectoration increases enormously, the wasting is marked and gurgling rales are present. If the possibility of the disease be remembered, pneumonic phthisis will not remain long unrecognized.

(b) **PULMONARY INFARCT.**—A source may generally be found for the embolus causing the infarction. In a recent case, three separate infarctions, each with a distinct history, were present in the lungs at one time, the source of the emboli being an inflamed varicose vein of the left leg. Circumscribed dulness and rales are often present. Bloody expectoration, rather than the rusty, jelly-like variety seen in pneumonia, the absence of preliminary chill, of typical fever and of microscopical evidence of pneumonia in the sputum are to be expected. If the embolus be a septic one, a true pneumonia, followed by abscess or gangrene may occur.

(c) **PULMONARY CONGESTION.**—In its acute form this may give the signs of and even the symptoms of acute pneumonia, being in fact the first stage of the latter disease. Only the failure of an acute pneumonia to develop enables us to make a diagnosis. In the chronic form, pulmonary hypostasis, there is a cause involving a weakened circulation, and long recumbent posture, as in typhoid, or in the chronic diseases. The signs are found in both bases behind. Acute pneumonia may develop upon this basis.

(d) **PULMONARY EDEMA.**—The presence of a cause for it, such as valvular disease, chronic nephritis, or high arterial tension, absence of typical history and of temperature, thin frothy character of the sputum, and the presence of fine rales in the dependent portions, and the lack of marked dulness and of bronchial respiration should suffice. The type of acute pulmonary edema with pink frothy expectoration, found in mitral stenosis, of which I have seen several examples, could scarcely be confounded with a febrile disease. The collateral edema in the unaffected lung in severe pneumonia should be mentioned.

(e) **PLEURAL EFFUSION.**—Attention should be given to the history, showing a milder onset, less degree of fever, less expectoration,



less severe prostration, and lack of crisis. The effusion gives rise to a greater dulness or flatness (if we except the comparatively rare massive pneumonia), to absence of fremitus and rales, diminished voice sounds and to restricted respiratory movements. Careful percussion will show displacement of neighboring organs, and often obliteration of Traube's semilunar space upon the left, if the effusion be a considerable one. Bulging of the intercostal spaces and increase of the circumference of the side of the chest involved are present. The S-shaped upper border of the dulness and the movable line of dulness in certain cases upon change of position, are to be considered.

Aspiration is so certain and so harmless a procedure that it should always be adopted in case of doubt. A needle of fair caliber and length, decidedly larger than the ordinary hypodermic needle, should be inserted at or near the center of the largest area of flatness, having due regard to the position of the heart. The finding of serum is conclusive. In interlobar exudates the diagnosis is much more obscure, and repeated punctures may be needed.

If the effusion be purulent there is a history of pneumonia in many cases, and the temperature will have been a septic one. Leukocytosis will arouse suspicion. The needle should be used. Grocco's sign is of less value than at first believed, since it may be detected in some degree in the absence of effusion.

(f) BRONCHOPNEUMONIA.—The lobar type of this disease gives rise to confusion in diagnosis. It is likely to occur in the very young, or, more often than is generally appreciated, in the debilitated and the aged. In children its frequent association with the infectious diseases should arouse suspicion. The occurrence of a precedent acute bronchitis, of irregular fever, commonly less severe than in lobar pneumonia, with absence of regular course and of crisis, of characteristic sputum, and of marked dulness associated with bronchial respiration, but with the presence of signs of acute bronchitis, and scattered patches of dulness and moist rales, and of a mucopurulent sputum, should suffice for the diagnosis.

(g) PNEUMOTYPHUS.—The diagnosis is impossible clinically until the signs of typhoid develop—rose spots, diarrhea, enlarged

spleen, etc. Laboratory examination may determine the diagnosis before these signs appear.

Meningitis and several other diseases might be considered but should offer no difficulty in the face of a careful physical exploration. No case of delirium tremens, acute mania or other serious disease should be passed without careful examination of the chest, which alone can prevent error. The frequency of acute pneumonia in pulmonary phthisis should be noted. There is grave danger, if one examine for the first time a patient known to have advanced tuberculosis, of overlooking the possibility of a complicating acute pneumonia, especially in the absence of an intelligent history of the onset. One then forms an exaggerated estimate of the gravity of the underlying tuberculosis, false because depending upon the signs given by the intercurrent disease.

**Prognosis.**—The mortality in Well's collection of nearly half a million cases was slightly over 20 per cent. The physician who states that he loses no cases of pneumonia has either no extensive experience with the disease, no power of observation, or no regard for truth.

The death rate is higher in cities, in the colored race, in the south than in the north, in the aged and infirm. Three-fourths of those past 60 die. It is graver in secondary types complicating influenza, etc., than as a primary disease. The percentage is commonly higher in hospital statistics and in figures taken from consultation practice than in strictly private practice, since broken-down material is largely the basis of the estimate in the hospital, and severe cases chiefly are seen by the consultant.

The death rate in the 250 cases quoted, all but 18 seen in consultation, and of grave type, was enormous—42.8 per cent. In this article I have stated the opinion that altitude is of no great influence in prognosis until it surpasses six or seven thousand feet. In cases of involvement of the entire left lung, in this series, but two recovered.

Certain families die so largely from this disease that a bad prognosis attaches to any attack of pneumonia, however mild. Marked leukopenia is of grave omen, 94 deaths occurring amongst

108 cases of pneumonia showing absence of leukocytosis in a total of 1,080 cases (Osler: "Modern Medicine"). Alcoholics are extremely bad risks. Patients with marked mitral stenosis commonly die within three or four days. I have known but one such to recover. Obesity is distinctly unfavorable. Pregnancy is a grave complication. Pneumococcic meningitis is practically always fatal. Collapse following the crisis is very serious. Emphysema, chronic bronchitis, tuberculosis of the lungs, pneumoconiosis, and chronic disease in general render the outlook grave. As I have shown elsewhere (*Denver Med. Times*, 1898) pneumonia is an extremely dangerous disease in case it attacks one isolated from assistance, because of the chance that delirium may come on and prevent his calling for aid. In sparsely settled countries many deaths occur amongst those so situated, several of which I have recorded.

Different years and different epidemics vary widely in mortality rate. Because of the fact that pneumonia is the terminal infection in many chronic diseases and in senility, there seems no good reason for expecting any improvement in regard to prognosis at all comparable to that which has taken place in many of the infectious diseases in recent years. The various sera introduced have as yet made no notable decrease in the mortality rate even in otherwise healthy individuals, but we may yet hope for assistance from this quarter.

#### IV. INFLUENZA

**Definition.**—La grippe occurs in pandemics more widely spread than those of any other disease. It is an acute infectious disease due to the bacillus of Pfeiffer. The widespread epidemic outbreaks have commonly been followed by minor local epidemics. Half the population of a city may be attacked within a few weeks. The spread of the epidemic westward during the invasion of 1889, along the lines of travel, was most striking. No immunity is conferred by an attack. A large proportion of the population is susceptible. The occasional escape of a prison or asylum community either wholly or for some weeks after the beginning of an epidemic goes to prove that exposure, not atmospheric or telluric influences, is necessary to infection.

**Symptoms.**—After an incubation period of one to three or four days, the disease begins with chilliness and malaise, only occasionally with a rigor. Fever, general pains, and muscular soreness appear, with astonishing prostration and weakness. In the milder cases, with the temperature of 101° or 102°, little follows but a coryza and a bronchitis, and the patient may not take to the bed. A case of average severity, however, presents more fever, more severe cough, chilliness, sore-throat, and sleeplessness.

**Varieties.**—(a) **RESPIRATORY FORM.**—This is the usual form in average epidemics, and corresponds closely to the description given in the last paragraph. The cough may be severe and in the old and feeble the infection frequently leads to bronchopneumonia or lobar pneumonia.

(b) **FEBRILE FORM.**—This is comparatively infrequent, and is of importance largely because of the need of differentiating it from typhoid, malaria, etc. It presents, like early typhoid, no distinctive features, there being simply fever, aching, prostration, furred tongue, chilliness, and perhaps slight nocturnal delirium. The failure to explain the attack upon any other hypothesis than that of the influenza is our chief ground for the diagnosis.

(c) **NERVOUS FORM.**—This shows but little of the catarrhal element of grippe; but headache, backache, sleeplessness, delirium, and prostration are notable.

(d) **GASTRO-INTESTINAL FORM.**—Watery diarrhea, nausea, vomiting, abdominal pain, prostration, and even collapse may be noted.

The varieties run so into each other that there is doubt as to whether it is best to try to classify cases in this manner. So long as one recognizes that influenza may manifest itself in other ways than as a "cold," the matter of classification is of little importance.

Leukocytosis, if present, is due to a complication. Smell and taste are often blunted. Herpes may be noted. Febrile albuminuria often occurs. After a course varying from one or two to ten days, convalescence begins, unless some serious complication has arisen. Relapse is common (10 per cent.).

**Complications and Sequelæ.**—The important complication is pneumonia, more often of the bronchopneumonic form. The death rate

from this cause in large cities often doubles during the prevalence of an epidemic. The young and old suffer chiefly.

The bronchitis of influenza at times becomes much more severe, with distressing cough, and eventually abundant sputum, often thin, but more characteristically greenish-yellow, and perhaps nummular. The severe types are those in which bronchopneumonia develops, and the complications ordinarily attending this disease are to be feared,—empyema, gangrene of lung, bronchial adenopathy, and pericarditis. The bronchiectasis developing years after an attack of grippe, a chronic bronchitis intervening, should be more widely recognized. The influenza bacillus is commonly associated with a mixed infection in these cases, and a dirty, offensive, and abundant expectoration occurs. I have had the dilated bronchi drained in two desperate cases, but both were shortly fatal. In this region, great numbers of tuberculous patients, often doing excellently, succumb either to some complication of influenza or because of a rapid development of the original disease as a result of its invasion.

The heart is greatly exhausted and depressed in many individuals attacked by influenza, and anginal attacks may occur. Endocardial and pericardial changes are less frequent than functional disturbances, such as intermittent heart, bradycardia, palpitation, etc.

The extension of the catarrhal inflammation to the sinuses and the middle ear is notoriously frequent, and antrum and mastoid operations are distinctly more frequently called for during epidemics of grippe. Deafness and vertigo often follow the ear complications and meningitis and brain abscess occasionally develop.

Neurasthenia, toxic peripheral neuritis, melancholia and insomnia are frequent sequelæ. The neuralgias so often mentioned should be recognized as such only after the exclusion of sinus involvement. Many other complications have been noted in this many-sided disease.

**Diagnosis.**—During an epidemic the patient commonly recognizes the disease before the physician is called. The great number affected and the vastly greater severity of the symptoms, especially the nervous ones, distinguish the influenzal outbreak from the common colds of winter. The finding of the bacillus of Pfeiffer in the bronchial

or nasal secretions is decisive, for it is not to be found in false influenza, or catarrhal fever, seen so often with or after epidemics of influenza vera, more especially with the minor endemic outbreaks than with the great pandemics. No causal organism is recognized in catarrhal fever.

From typhoid fever we should distinguish the febrile and gastro-intestinal types of gripe by the more sudden onset, much shorter course, absence of eruption and enlarged spleen, the finding of the Pfeiffer bacillus and the absence of the Widal reaction.

Cerebrospinal meningitis may be simulated and especially if an influenzal meningitis develops. The diagnosis must be made in the laboratory in obscure cases. Dengue has already been considered.

**Prognosis.**—Few cases die of influenza, excepting feeble infants and the aged. Many die from bronchopneumonia and other complications. The adults dying are among those suffering from chronic disease, which weakens the resistance to the attack, notably chronic nephritis, chronic bronchitis, myocardial disease, arteriosclerosis, and pulmonary tuberculosis. The evil influence upon the latter disease must not be forgotten. Many cases of chronic bronchitis originate in or are made worse by an attack of influenza, and bronchiectasis is not an infrequent sequel.

## V. DIPHTHERIA

**Definition.**—Diphtheria or angina maligna is an acute infectious disease caused by the Klebs-Löffler bacillus and characterized by a fibrinous exudate upon the mucous membranes of the upper air passages.

A somewhat similar membrane, indistinguishable clinically, may be due to the streptococcus and other organisms, and may be designated pseudo-diphtheria. So-called membranous croup is now classed as laryngeal diphtheria since in the great majority of cases it is due to the Klebs-Löffler bacillus.

Diphtheria is at first a local disease, and the constitutional symptoms result from the absorption of toxins generated at the

place of infection. In the larynx, especially, the disturbances may be chiefly mechanical. In severe diphtheria the advent of a secondary infection at the site of the membrane may add much to the severity of the disease.

**Varieties.**—Malignant diphtheria has extensive membranous deposit with the severest constitutional symptoms, and was always fatal before the advent of antitoxin. Tonsillar, pharyngeal, laryngeal, nasal, and conjunctival diphtheria are sufficiently definite forms to be recognized. The laryngeal form is often fatal from mechanical obstruction; the severe nasal form was nearly always fatal before the days of antitoxin. The membrane occasionally develops in the ears, vagina, upon the prepuce, or in open wounds.

The disease usually develops between the second and tenth years, but is not infrequent at any age below forty, and may occur later. Although depressing influences, overcrowding, etc., have a certain secondary influence, the essential feature in the production of diphtheria is infection with *Bacillus diphtheriæ*, resulting from its contact with a susceptible mucous membrane. Chronic disease of the tonsils, the presence of adenoids, and tonsillitis, pharyngitis, measles, scarlet fever, grippe, etc., all predispose to the development of diphtheria. It is decidedly more frequent in cold weather. Previous attacks predispose to the infection. Natural immunity is comparatively common, and physicians rarely contract the disease unless under conditions of especially severe exposure, or of direct infection, as from coughing on the part of the patient. Adler states that the insane rarely show typical clinical diphtheria.

The presence of the *B. diphtheriæ* in the throat in those who have recovered from the disease and even in those who have never had an attack is important. These "carriers" have been responsible for the spread of the disease in many instances. Self-infection may occur in the carrier himself.

**Transmission.**—The disease is commonly transmitted by the conveyance of the bacillus from the lesions of the bearer to the receiver's mucous membrane, as in coughing, kissing, using the same utensils, towels, drinking cup, pencil, etc. The saliva is laden with the bacilli and laryngeal cases in which coughing occurs and in

which, therefore, droplets of sputum are widely scattered, are especially dangerous to others. The lower animals may contract and spread the disease directly, or carry infection by means of their fur, hair, etc. Cats and fowls are especially susceptible to diphtheria. A pet antelope contracted it in one instance under my notice. The disease has been spread in some cases through milk.

**Incubation.**—This period is from one to three days, or longer in some cases. Then, without especial prodromata generally, the usual signs of acute infection develop,—malaise, mild fever, perhaps in children convulsions, but these phenomena vary greatly. If old enough the patient complains of sore-throat, and dyspnea. A preliminary erythema may be noted. Examination of the pharynx and tonsils now commonly shows marked congestion, sometimes of a purplish hue. Within the next 12 to 24 hours patches of dirty white membrane appear, especially at first upon the tonsils, and spreading thence to the uvula and soft palate, and perhaps to the nares and larynx. The membrane may be absent or may merely fill the crypts of the tonsil, giving the appearance of an ordinary lacunar tonsillitis.

In the earliest stages the membrane may be removed without great difficulty, but when fairly developed a bleeding surface remains. If the membrane spread but little beyond the tonsils and nearby structures, the case may be mild, and the patient may not even see a physician. Many epidemics originate from such unsuspected cases. The development of a post-diphtheritic paralysis or nephritis may lead to a correct diagnosis weeks afterward.

A foul odor is given off ("putrid sore-throat") as the membrane develops. The spread of the membrane is more rapid if the mucous membrane be damaged by too strong gargles or applications, for the bacilli cannot grow upon a normal mucosa. The pulse is increased in frequency at first, and by the second day is likely to be soft, weak, and rapid. The temperature is commonly not over 102° to 103° F., a higher temperature depending upon a mixed infection or some complication.

We have spoken especially of tonsillar and pharyngeal diphtheria of the ordinary grade. In the severer forms of the latter, the lymph



nodes enlarge and become tender, and there is a tendency for the membrane to spread further.

**Nasal Form.**—The membrane extends to the posterior nares in many cases, generally of a severe type, but occasionally mild forms originate here, giving rise to the symptoms of a coryza. There is danger, however, of spread to the sinuses, through the lacrimal ducts to the conjunctivæ, through the Eustachian tube to the ear, and bronchopneumonia is especially likely to develop as a complication. Because of the danger of spreading diphtheria, patients with a nasal discharge, which excoriates the nares, should be examined for the bacillus, for many such, without constitutional symptoms, have been found to carry the bacillus. Some surgeons go to the point of always taking a culture from the throat before any operative measures upon the throat or palate, for fear of infection of the wound by a latent diphtheritic infection.

**Laryngeal Diphtheria.**—This may or may not show membrane in the larynx, and the absence of such a finding does not count against the diagnosis. A croupy metallic cough, aphonia, or harsh voice, dyspnea, or progressive laryngeal stenosis, are to be noted. Cyanosis soon develops. Death may result from toxemia and exhaustion, from bronchopneumonia, either from spread of the membrane downward or, more commonly, from inhalation of the infective material, or from gradual blocking of the larynx from growth of the membrane, or sudden blocking of the larynx by a detached fragment of membrane. An actual diphtheria of the lung may be found post mortem. Substernal and supraclavicular retraction, orthopnea, and dilatation of the alæ nasi are prominent features. Rigidity of the sternomastoid muscles is ominous. The mortality is highest in this type of diphtheria, though occasionally cases recovered, even in pre-antitoxin days.

Diphtheria in other situations may cause post-diphtheritic paralysis, nephritis, etc., but it is not commonly fatal. The eye may be lost, the prepuce may slough, the female genitalia may be severely swollen, with marked abdominal symptoms, and death may occur in either form. Wounds of the fingers in intubation are decidedly serious. Any of these infections may be autogenous from the pa-

tient's own nasal discharge, perhaps unsuspected. The anus is rarely infected. Diphtheritic membrane has been found in the stomach and duodenum.

**Complications.**—These are numerous and severe. Bronchopneumonia is probably the most frequently fatal. Otitis media is common. Gangrene of lung is rare. Acute nephritis is less frequent than with scarlet fever and less frequently accompanied with edema. A toxic albuminuria is to be expected in all severe cases. The circulatory complications are important. Endocarditis and pericarditis are unusual, but a toxic myocarditis is frequently seen, with dilatation, relative leakage, notably at the mitral orifice, irregular heart action, epigastric distress, and death in a large proportion of the cases.

Paralysis as a sequel occurs in the second or third week in 15 per cent. to 40 per cent. of all cases, though rarely recognized in such a proportion unless carefully sought for. There may be nothing more than an absence of the knee-jerks, which should suggest further examination of the nervous system. Paralysis of the uvula and of the intrinsic and extrinsic muscles of the eye may escape notice, although double vision, loss of accommodation, and ptosis may call attention to it later.

Palatal paralysis is probably the most frequent form, characterized by nasal voice, difficulty in swallowing, and regurgitation of food through the nose. Paralysis of the arms and legs, of the respiratory muscles, and, a little later, general multiple neuritis may develop. An acute ataxia has been noted, attributed to the action of the toxin upon the posterior nerve roots and posterior columns of the cord.

The most serious neuritic complication is the vagus paralysis, which indeed may be the underlying, but unrecognized, feature in certain fatal cases attributed to myocardial disease. Increased frequency of the pulse, or more serious still, marked slowing of the rhythm, with irregularity, dilatation of the heart, valvular murmurs, prostration, vomiting, and death may be seen. Continued vomiting with prostration and feeble pulse, either fast or slow, is of gravest omen, signifying vagus involvement. Death from diphtheria after

three weeks have passed is generally chargeable to this dreaded complication.

Cervical adenitis, even with suppuration, may occur, the latter due to mixed infection. Serum urticaria and other rashes may occur. Petechiæ may develop in grave cases. Epistaxis may be severe. Mastoid involvement is not very common.

A moderate leukocytosis is present in diphtheria and increases with septic complications. A post-diphtheritic anemia is very common.

**Laboratory Diagnosis.**—This concerns especially the detection of the Klebs-Löffler bacillus and its differentiation from the pseudo-bacillus. A bullion culture of the latter fails to kill a rabbit or guinea pig in 24 to 48 hours, while that of the true organism is fatal. In case of dispute this test should be applied.

**Diagnosis.**—**DIRECT.**—This rests on the acute illness as described, with false membrane and demonstration of the Klebs-Löffler bacillus. Yet the two former features may be absent without in any way lessening the dangers to the community from the patient. Further, the patient may have acute illness and the membrane, but the laboratory may show it to be due to the streptococcus or the pseudodiphtheria bacillus (*B. xerosis*).

Streptococcic angina is likely to be more severe in onset, to have a higher temperature, and to have more pain. Septic, especially suppurative, complications are more frequent. The diagnosis is settled in the laboratory.

The staphylococcus pyogenes aureus was frequently found with the diphtheria bacillus in McCollum's experience. In 60 nurses examined by him in the South Department of the Boston City Hospital no positive culture was obtained, so that he doubts the frequent presence of the diphtheria bacillus in the throats of healthy persons regardless of the degree of exposure.

The presence of the diphtheria bacillus in the throats of patients with measles and scarlet fever has been mentioned. All patients with throat symptoms and exudate should be examined culturally regardless of the clinical aspects of the affection.

In case of doubt or impossibility of laboratory aid the diagnosis

is occasionally cleared by the miraculous effect of the antitoxin, which is without effect in any but cases of true diphtheria.

**Prognosis.**—The average death rate, previous to the introduction of antitoxin was from 30 per cent. to 50 per cent. I have seen it 100 per cent. in small epidemics. This rate has fallen with earlier and more common use of the serum to less than 10 per cent. In States sparsely settled and of great area, with few laboratory facilities and difficulty in communication, the rate is higher, and is definitely higher in the outlying communities than in cities. The rate for 5,388 cases in 1901 to 1904 in Colorado was 16 per cent., this being unduly high because of the above-mentioned features. I have seen but one death when the antitoxin was administered in proper dose within the first 24 hours of the disease.

## VI. VINCENT'S ANGINA

Recently cases of angina with ulcerative stomatitis have been described, occurring in children, and associated with the two organisms described by Vincent, a spirochete and a fusiform bacillus. Cases clinically resembling these have been described, however, in which the organisms were not found.

The disease develops as an ordinary angina, often with severe constitutional symptoms. The tonsils are generally first affected, but the ulcerative and perhaps membranous deposit often extends to neighboring parts, and even to the pharynx and gums. The exudate is soft, grayish-yellow or greenish, easily detachable, and leaving a bleeding, slightly depressed ulcerative surface. Extensive destruction, especially of the soft parts, may occur. The diagnosis is made in the laboratory, diphtheria and syphilitic mucous patches being often suspected.

**Prognosis.**—This is generally good, patients recovering in five to twenty days in average cases.

## VII. ERYSIPELAS

**Definition.**—This is an acute infectious disease caused by the streptococcus, transmissible under certain conditions, and charac-

terized by a definite inflammatory condition of the skin and subcuticular tissues.

**Etiology.**—The causative organism is now regarded as being identical with the ordinary streptococcus pyogenes, and the infection of man from streptococcic sepsis may produce typical erysipelas.

Anders found that 19½ per cent. of 2,010 cases occurred in April, and 50 per cent. during February, March, April, and May. Males of the third decade suffer somewhat more often than other individuals, but neither race, age, nor sex is of extreme importance excepting as offering opportunity for the infection.

The mode of infection is through inoculation of an abraded surface with the streptococcus derived from a case of erysipelas or other streptococcic process. The mucous membrane of the nose is a frequent site of origin. In many cases a wound or abrasion elsewhere becomes infected. Puerperal conditions offer many opportunities for infection unless the most scrupulous care is used by the attendant. Instruments and unclean hands frequently carry the disease. Careless vaccination may be the cause. I knew a physician who had himself had six attacks of facial erysipelas immediately after examining cases of the same disease; I was impressed with the freedom with which he handled the infected area, and the neglect of antiseptic precautions afterward, and I do not doubt that he infected his nasal mucous membrane or other surface presenting some minute abrasion by means of his own fingers. A personal predisposition presumably existed.

Recent wounds, recent confinement, debilitating causes (alcoholism, senility, chronic diseases), and general unhygienic conditions favor the development of erysipelas. One attack predisposes to another.

**Incubation.**—This period probably varies between twenty-four hours and a week, perhaps generally two to four days. Infection of a wound often results in the prompt development of the disease, while in the absence of a recognized abrasion several days may elapse.

**Symptoms.**—Without definite prodromata, or with headache, slight fever, and malaise, the patient is seized with rigor, perhaps only chilliness, vomiting, and a rapid rise of temperature to 104° or

105° F. No especial pain is experienced, but a feeling of burning and tension at the involved area. Great prostration develops shortly in the feeble. A full rapid pulse and leukocytosis are present.

Within a few hours a reddened spot is found, upon or near the nose in a majority of cases, swollen, tense, edematous, and shining, and sharply delimited, with a definitely raised border. Small vesicles or bullæ may be noted. The process spreads rapidly, tending to involve the whole face and scalp. When fully developed the swollen face, closed eyes, deformed ears, and obstructed nostrils often render the patient wholly unrecognizable.

Swelling of the lymphatic glands occurs. The mucous membrane of the mouth and throat is involved, and the larynx may be obstructed by edematous infiltration. Small abscesses may be present. The mind is often clear, but nocturnal delirium is of frequent occurrence. In the severer types complicated with deep-seated suppuration, pneumonia, endocarditis, or other septic complication may develop, and violent delirium may be noted. Moderate splenic enlargement, rapid pulse, febrile urine, albuminuria, coated tongue, and constipation are commonly present.

After three or four days the inflammation at a given point reaches its acme, and begins to subside, while the process extends at the margins. After six or seven days, in favorable cases, the fever subsides, often by crisis, and the inflammation ceases to extend. If new areas become involved, lesser febrile developments follow. Definite relapse is rare.

Sub-varieties are recognized as follows:

(a) *ERYSIPELAS MIGRANS*.—The acute process tends to become subacute and may spread over the whole body. The disease may last six to twelve weeks. I have seen the same area “burned over” a second time. Death from exhaustion is common in the feeble and in infants. These patients seem unable to develop an antitoxin to the disease.

(b) *E. PHLEGMONOSUM*.—Deep suppuration may occur in the subcutaneous tissue, and the muscles, periosteum, and even bone may be involved. The gangrenous form is an advanced stage of the phlegmonous variety. I have known more than a dozen large in-

cisions required in the face, with recovery, but with frightful scarring.

(c) *E. NEONATORUM*.—This form follows infection at the navel in the new-born. It spreads rapidly, and is commonly fatal in a few days.

Relapsing erysipelas has a notable tendency to recur. In certain forms, especially in the debilitated, but little fever is present. Hyperpyrexia, with coma and death, is not infrequent. With acute suppuration of the middle ear I have seen erysipelas of the usual type develop, and with the chronic form, repeated circumscribed mild attacks, distributed over several years, and confined practically to the ear and the surrounding tissues.

**Complications and Sequelæ.**—Anders found in 1,674 cases abscess 105 times, arthritis 20 times, delirium 10 times, lobar pneumonia, phlebitis and pleurisy each 7 times, nephritis 6 times, synovitis and diarrhea each 5 times, tonsillitis 3 times, and other complications occasionally. Endocarditis and the whole train of septic diseases may be noted. The lungs and the kidneys may be involved, the tissues being infiltrated with the streptococci. Functional heart murmurs are frequent. Lupus, chronic eczema, and even sarcoma are favorably influenced by the erysipelatous process in certain cases.

**Diagnosis.**—The acute onset, fever, sharply defined inflammatory process, a tendency to spread, with decline after three or four days at the point of original infection, and the finding of the streptococcus, often associated with the staphylococcus and a diplococcus, in the serum from the infected area, commonly establish a diagnosis without difficulty. In erythema, notably erythema nodosum, in urticaria and acute eczema, the absence of signs of an acute infectious febrile process are of the greatest value. It is fairly safe to exclude erysipelas in the absence of fever, but this may not exceed 1° F. The anthrax pustule presents a black center, and the bacteriological differentiation is easy.

**Prognosis.**—Anders found the death rate to be 6.57 per cent. in the hospital cases and 4.17 per cent. in private practice, and these rates accord well with general experience. In infants and the aged the rate is high. Adults commonly recover, unless chronic dis-

ease, alcoholism, or debility from other cause is present. Death results from the intensity of the infection, from septic complications, or from exhaustion. The introduction of antisepsis in surgery and in the lying-in room has done much to decrease the incidence of erysipelas and the mortality rate is less than formerly.

### VIII. CEREBROSPINAL MENINGITIS

**Definition.**—Epidemic cerebrospinal fever is an acute infectious disease due to the meningococcus (*diplococcus intracellularis meningitidis*) with especial localization in the meninges of the brain and cord.

The disease is found in temperate climates, in youth chiefly, but occasionally at all ages and at any season, though rather more frequent in winter and spring. It is especially found under conditions of overcrowding and uncleanness and rather more often in the city than in the country. Soldiers in barracks and inmates of badly arranged and ventilated sleeping quarters are frequently attacked. The mode of conveyance is still under investigation. Individual immunity must be common, for we rarely see more than one case in a family or household. There seems to be no proof that it is ever carried by a third person.

It is to be assumed that the disease is carried by the secretions of the mouth, nose, and eyes, for the meningococcus has been repeatedly demonstrated in these secretions. The disease is not contagious in the sense that measles and scarlet fever are. Immunity is commonly established by one attack, but a second may occur.

**Clinical Forms.**—The disease varies immensely in different individuals and in different epidemics.

(a) **ORDINARY TYPE.**—After an unknown period of incubation the disease begins abruptly, with chill, vomiting, and violent headache, and backache, photophobia, delirium, coma and convulsions may supervene. Convulsions may be among the earliest features in children. Vertigo may replace or accompany headache. Hyperacusis, hyperesthesia, and priapism are common. Pains along the spine and in the extremities appear, and stiffness and retraction of the neck,



pain upon swallowing and upon moving the spine, strabismus, ptosis, nystagmus, irregularity of the pupils, facial palsies, opisthotonus, muscle cramps, and hemiplegia, paraplegia, or other types of paralysis are common features. The patient is restless and irritable, and a violent delirium may occur, often followed by somnolence or coma.

The temperature runs, ordinarily, between 100° and 103° or 104° F., and often higher before death. It is more irregular and less characteristic than in most acute infectious diseases. The pulse, full at first, is likely to become feeble, soft, weak, and irregular, and of moderate frequency. The slow pulse noted in tuberculous meningitis is not often seen. The respiration is apt to be irregular, sighing, and moderately increased in frequency. Cheyne-Stokes respiration is common. The tongue resembles that of typhoid. Constipation is the rule. Polynucleosis of 20 to 55 thousand occurs. The spleen is often moderately enlarged. The urine is generally albuminous and may contain blood. Traces of sugar are not infrequently present. Retention and polyuria are occasionally seen.

Herpes is frequently noted, and may be valuable in diagnosis. The eruption may not be seen in certain epidemics, and yet may be a most prominent feature in others. Small petechiæ are most common, but large ecchymoses may develop (spotted fever), especially in the malignant type of the disease. Erythema is most common, but a roseolous eruption, urticarial mottling of the skin, and lesions like those of erythema nodosum may develop. Pemphigus and gangrene have been recorded.

*Eye Lesions.*—Paralysis of ocular muscles is frequent and of great value in diagnosis. The oculomotor nerve is frequently compressed by the exudate at the base, and the fourth and sixth nerves may be involved. The inflammation may extend along the optic nerve into the orbit, with complete destruction of the eye. Conjunctivitis may occur either as a result of a specific infection by the organism of the disease, or through loss of the protective influence of the fifth nerve when it is involved by a neuritis. In certain epidemics blindness has been a common sequel.

*Ear Lesions.*—In children especially auditory involvement is

very common, and many of the cases of deaf-mutism in certain irregularly distributed periods have been due to the disease. Deafness may result from involvement of the auditory nerve in the exudate, with subsequent degeneration of the fibers, especially in the chronic cases, or from disease of the labyrinth. Otitis media, with presence of the diplococcus in the pus cells, is not infrequent. Early acquired deafness is attributed by many otologists to abortive attacks of cerebrospinal meningitis. Over half the cases recovering in many epidemics are deaf, though the deafness may not become marked for weeks or months after the convalescence.

(b) **MALIGNANT FORM (Meningitis Siderans).**—This differs from the ordinary form in its extreme rapidity and severity. I have known death to occur in eight or nine hours. The patient is stricken with violent chill, vomiting, headache, moderate fever, perhaps with convulsions, cyanosis, delirium, coma, purpuric eruption, and death in rapid succession. These cases are more common at the beginning of an epidemic and in certain seasons.

(c) **ABORTIVE FORM.**—It comes on severely, but subsides in two or three days. The occurrence of deafness as a result has been noted.

(d) **INTERMITTENT FORM.**—In this variety probably owing to successive involvement of new foci, but perhaps to renewed growth of the diplococcus, the symptoms become more severe every two or three days, subsiding between times. The temperature is irregularly remittent.

(e) **MILD FORM.**—Diagnosis of this variety is possible only during epidemics, unless by spinal puncture. Fever may be trivial or absent. Headache, malaise, stiffness of the neck, nausea, vomiting, and vertigo may be noted in various combinations. In a physician's wife I noted headache and slight rigidity of the neck, with an intolerable hyperesthesia, requiring morphin hypodermically for over a week, with complete recovery.

(f) **CHRONIC FORM.**—These cases, instead of recovering, as in the ordinary forms, during the first month, may run on for two to five months, with extreme emaciation. Exacerbations and remissions occur, and the patients may die of exhaustion or the complications

of the disease, or finally recover. Chronic hydrocephalus is probably active in producing the symptoms in certain cases, and abscess of the brain, general neuritis, or other post-meningitic lesions doubtless explain others.

**Complications and Sequelæ.**—The eye and ear complications have been mentioned. Pneumonia, which may be due to the specific organism of meningitis, may occur, especially toward the end of an epidemic. Suppurative arthritis is common in certain epidemics, but milder forms of joint involvement are much more often seen. Meningococcus septicemia may accompany the joint trouble, the endocardium being generally involved. Parotitis, sometimes suppurative, pericarditis, endocarditis, and nephritis may occur. Elser found the meningococcus in the blood in about 25 per cent. of the cases examined.

The sequelæ are commonly the direct results of the damage done to the meninges and the brain by the inflammatory process, or of a peripheral neuritis. Hydrocephalus, chronic headaches, idiocy, or other forms of mental impairment, damage to sight, speech, hearing, taste, paralysis of any of the cranial nerves, paraplegia, etc., are not infrequent.

**Diagnosis.**—The recognition of cerebrospinal fever depends upon the presence of definite meningeal signs and symptoms, for the phenomena due to the severe general infection are not sufficient for diagnosis from other infectious diseases. If there are added to the general features of a severe infection such as might occur in variola, typhus, pneumonia, scarlet fever, or meningeal typhoid, Kernig's sign, deafness, blindness, ptosis, ocular paralysis or paralysis of other muscles, the diagnosis becomes presumptive, and may ordinarily be established by lumbar puncture and examination of the exudate.

Kernig's sign is present in 85 per cent. or so of cases of meningitis, and adds much to the probability of the diagnosis, although it may be found in the cases of so-called meningeal typhoid, and may be absent in the malignant, comatose type of meningitis. The discomfort and often quite sharp pain should be manifested when, in the attempt to straighten the leg upon the flexed thigh, an angle of

90° to 135° is attained. A strong contraction of the flexors of the thigh appears.

**LUMBAR PUNCTURE.**—Quincke's method has added so much to our ability to diagnose meningitis that it should be adopted early in doubtful cases. The generally turbid exudate contains the intracellular diplococcus and often many of the cocci free in the fluid as well. It is necessary to withhold the diagnosis and repeat the procedure later if the result be negative. Koplik finds that in the absence of both the meningococcus and the tubercle bacillus, cerebrospinal fever is more probable in any given case, since tubercle bacilli are found with more certainty in the tuberculous meningitis than the diplococci in chronic spinal meningitis.

The advisability of being prepared to use the Flexner serum if possible through the same needle, if the fluid is turbid, should be mentioned.

**Differential Diagnosis.**—We may generally distinguish tuberculous meningitis by its prodromal period, more gradual onset, asthenic course, absence of petechiæ, of hyperesthesia, less degree of leukocytosis, delay in appearance of ocular paralyses, slow and irregular pulse, and, finally, by the results of the puncture.

Pneumococcic meningitis is often associated with pneumonia or with ear trouble, and in general lacks the sharpness of outline of the picture of the epidemic form. The cranial nerve signs are generally less marked. A temperature of 108° to 109° F. has preceded death in several of my cases, and every one has been fatal.

Streptococcic meningitis is secondary to traumatism or infection elsewhere, and is much more frequent after the streptococcic throat infection than is commonly recognized. Erysipelas, endocarditis, otitis media, and general streptococcic septicemia are common sources. The meningeal signs are often less prominent than those of the general infection.

The distinction between epidemic meningitis and infectious diseases in general, notably meningeal typhoid, typhus, and scarlet fever has been discussed under the different diseases mentioned.

Posterior basic meningitis, of Gee and Barlow, is simply the form of cerebrospinal meningitis occurring in children under two

years of age. The meningeal process is limited chiefly to the posterior aspect of the base of the brain, and the striking sign is the "holding back of the head" (cervical opisthotonus, mentioned in the early description of the disease). The finding of the diplococcus intracellularis meningitidis has settled the identity of the disease.

A similar limitation of the meningitic process, with the cervical sign mentioned, is possible in streptococcic meningitis, but no proof is offered that any basic meningitis exists separate in etiology from the forms of meningitis with the usual distribution.



FIG. 29.—CEREBROSPINAL MENINGITIS, BASILAR TYPE. Hydrocephalus. Chronic course. Proved bacteriologically to be of the epidemic form.

**Prognosis.**—The severity varies extraordinarily in the different epidemics, from 20 to even 70 or 80 per cent. being fatal. Adults offer a better prognosis than children. The early use of the Flexner serum greatly improves the chances for recovery.

## IX. PERTUSSIS

**Definition.**—Whooping-cough is an epidemic disease characterized by a peculiar convulsive cough, the paroxysm ending with an inspiratory "whoop."

Nearly everyone exposed, regardless of the time of life, if not immune through a previous attack, contracts the disease. It is never-

theless chiefly an affection of the first two or three years, because of the almost inevitable exposure early in childhood. It is much more prevalent in the colder months, and feeble individuals contract the disease more easily than others. The contagion doubtless resides in the sputum and mucous discharges of the nose and throat, since the disease may be contracted by associating with those suffering even with the early catarrhal stages, or from exposure to fomites (handkerchief). The Bordet-Gengou bacillus is finally recognized as the causative agent. Whooping-cough is one of the most difficult of diseases to control, since it is infectious days or weeks before the appearance of the characteristic whoop upon which the diagnosis depends.

The occurrence of epidemics every two or three years in cities, and the association with epidemics of measles, scarlet fever, and chicken-pox, should be noted.

The period of incubation varies between seven and ten days. A catarrhal stage with coryza and ordinary or severe bronchial cough gives way after eight or ten days to the paroxysmal stage in which the "whoop" appears, while the slight fever disappears. A sensation of tickling in the region of the larynx and trachea is noticed, and the child may run to cling to its mother's dress, anticipating the onset of the paroxysm. A crying spell, or other trivial cause often starts the attack. After a long inspiration, perhaps a dozen short coughs follow, of such intensity and rapidity that inspiration is impossible. Then comes a long inspiration with such difficulty of entrance of the air to the larynx that the characteristic whoop is produced. An abundant discharge of tenacious mucus may occur at this time, with nasal discharge also, or one or two further attacks may follow in rapid succession before such relief is attained.

During the paroxysm the face becomes cyanotic, the eyeballs project, and the veins of the neck swell. Involuntary micturition during the height of the paroxysm is frequent and loss of control of the sphincter ani may be present. The individual attack lasts a quarter to a half minute, but successive paroxysms may prolong the time to several minutes, leaving the child relaxed, perspiring, and exhausted. Occasionally a vein in the loose tissues about the orbit or conjunctiva

is ruptured and extensive ecchymosis appears. In the prolonged and severe attacks seen in feeble children, often complicated with a bronchopneumonia, the bulbus jugularis becomes so distended that the venous tumor resulting above the right clavicle causes anxiety not only to the parents, but to the physician, if he be not familiar with the extent of the distention which may occur.

The paroxysm may be excited by anger, laughter, attempts to eat or drink, and especially by efforts to examine the throat. Vomiting so frequently occurs as the result of the attack that the child loses weight and strength rapidly, and care should be taken to give food as soon as possible after the cough.

The chafing of the engorged tongue upon the incisor teeth often causes abrasion and ulceration of the frenum, a mark by which the disease may sometimes be recognized when other evidence is not available. Examination of the chest shows little except rough breathing with occasional dry rales such as might be found in a mild bronchitis. Redness and occasionally ecchymosis or superficial ulceration may be found about the larynx, if the physician be sufficiently expert to examine it without inducing an attack. A plug of thick mucus characteristic of the disease has been seen in the trachea, and the accumulation of this mucus seems to be intimately related to the production of the attack.

The paroxysms number a dozen to twenty in 24 hours in the ordinary type of whooping-cough, but may be but three or four, or in extremely severe types even 100. They are more frequent in the night and indoors than otherwise. The "whoop" may be absent.

In the average cases the paroxysms become less severe after three or four weeks and disappear a week later, the catarrhal bronchitis persisting for some weeks. In cold climates, the child may cough for the remainder of the winter despite every attention. The entire course of the disease is thus likely to be two months in even moderately severe cases and three or four in others. In the later stages of the cough the infectivity of the disease is absent, although no one can determine the exact time. The continued cough seen in some cases with exacerbations upon the slightest exposure has been called a "habit cough." It is probable that in most cases the well-recog-

nized enlargement of the tracheobronchial glands, occasionally demonstrable by physical examination, constitutes the basis of this variety of cough. Relapse is rarely seen in whooping-cough, though second attacks may occur in later years.

**Complications.**—The most serious of these is bronchopneumonia, which is the common cause of death when this disease is fatal. I have seen asthma of great severity as a sequel in the adult. Chronic bronchitis, perhaps with emphysema, lobar pneumonia, pleurisy, and nephritis may be noted. Tuberculosis is frequent enough to demand anxious supervision of any patient not recovering normally. The enlargement of the bronchial glands mentioned as the cause of persistent cough is so constant and important a feature of whooping-cough that it deserves closer attention than is commonly given it. A leukocytosis of 20,000 or 25,000 with notable increase in the lymphocytes is present. The false diagnosis of lymphoid leukemia is occasionally made because of the latter feature.

Other complications are mechanical in nature, from the great increase in the blood pressure in the venous system during the paroxysms, from the increase of air-pressure in the respiratory tract, or the intra-abdominal pressure. From the first cause we may have the ecchymoses alluded to, slight hemorrhages from the ear, nose, or bowel and even meningeal or cerebral hemorrhage, with convulsions, aphasia, hemiplegia, or sudden death in the rarest instances. Dilation of the heart, especially the right side, is common. If valvular leakage should occur it is much more likely to be relative than organic, as endocarditis is a rare sequence. Irregularity of the pulse is often noted, especially just after the paroxysm.

From the second cause may arise interstitial emphysema, when actual rupture of an alveolar wall or small bronchus occurs, with extension to the mediastinum or the subcutaneous tissues of the neck. I have known it to extend to the fingers. Acute emphysema, usually transient, is not infrequent, and pneumothorax may occur.

The violent abdominal strain may cause hernia, prolapse of the bowel, and uterine prolapse. Abortion is to be feared in case a pregnant woman suffers from a severe attack.

It is impossible to be sure of the diagnosis of whooping-cough



until the "whoop" appears. A cough gradually increasing in severity, worse at night, with congested eyes, suffused face, and vomiting may be assumed to be pertussis even in the absence of the diagnostic feature, and if there be a history of exposure to the disease, the presumption becomes almost a certainty. The imitative cough seen in school children should be mentioned, and the hard ringing cough, less distinctly paroxysmal, and without "whoop," vomiting or expectoration, associated with enlarged tracheobronchial glands. The bacteriological diagnosis is not yet upon a satisfactory basis.

**Prognosis.**—This is good in mild cases without complications. During the first year and to a less extent in the second, the incidence of bronchopneumonia gives rise to the chief mortality of the disease, and the rate may rise to 6 per cent. or 8 per cent., or even much higher amongst the feeble children in the foundling asylums, etc. Such infants, and the aged and debilitated furnish the material from which most of the fatal cases come. The mortality is greater in the colored race and in the winter.

The mortality of whooping cough cannot be as exactly estimated by means of health office statistics as that of the reportable diseases. That it is a serious disease, however, is shown by the comparison, during the four years, 1901–1904, of the deaths reported to the Colorado State Board of Health. From whooping-cough there were 162, from measles 178, from scarlet fever 618, and from diphtheria 887.

## X. MALTA FEVER

**Definition.**—Malta or Mediterranean fever is an acute infectious disease caused by the micrococcus melitensis, and characterized by long-continued and irregular fever, with apyretic periods (undulant fever). The association of the disease with the keeping of goats and the use of the milk is emphasized by the occurrence of recent cases in Texas.\* It is likely that more cases will be detected upon investigation, more especially because the goat, although a carrier of the infection, remains in apparent health and continues to give milk.

**Etiology.**—The disease prevails extensively upon the shores of

\* Gentry and Ferenpaugh: *Jour. Am. Med. Assoc.*, Sept. 9, 1911.

the Mediterranean and its islands, and occasionally in the West Indies, in China and in India. The disease is transmitted by the specific organism found to be present in the milk of the infected goat. The micrococcus is also present in the blood and urine of the goat, and of patients suffering with the disease. There is no basis for the theory that the mosquito may transmit the disease, and it is not considered transmissible amongst the patients in hospital wards. The specific organism is found abundantly in the spleen in life and after death. The young adults of the British army in Mediterranean stations have been the greatest sufferers from Malta fever. The disease is especially prevalent in summer.

**Symptoms.**—The period of incubation is believed to be between a few days and three or four weeks, but is not definitely known. Probably six to ten days would be an average time.

Prodromes similar to those of typhoid are often noted. The characteristic feature of the disease is the so-called “undulant fever.” The pyrexia reaches a height of  $103^{\circ}$ – $104^{\circ}$  F., persists for one, two, or three weeks, subsides or remains normal or nearly so for two or three days to two weeks, rises again, and continues in this way for an average period of about three months. This may be extended to six months and with relapses, which are fairly frequent, to even two years.

Constipation is the rule, but diarrhea may occur and the stools may show a little blood. Sweating, arthritis, in some cases with effusion, enlargement and tenderness of the spleen, progressive anemia, and marked debility are characteristic features. A malignant type, fatal in eight or ten days, is occasionally noted.

The polymorphonuclear cells of the blood are moderately increased. The micrococcus *melitensis* gives a characteristic agglutinative reaction, a complement-fixation test has been developed, and blood cultures have of late been positive.

**Diagnosis.**—In the endemic areas the diagnosis is easy. In the isolated cases, seen elsewhere, diagnosis would be practically impossible in the absence of exposure in the areas of infection, excepting by means of the laboratory procedures mentioned. Malaria and typhoid present no leukocytosis, but the finding of the parasites in

the former and the Widal reaction in the latter are conclusive. The long continuance of the fever, in the absence of known cause, and the soreness about the joints, and especially the association in some manner with goats or their milk, should give rise to the suspicion.

**Prognosis.**—The English have found the mortality rate to be about two per cent. Recovery is more speedy and sure upon sending the patients away from the region of infection.

## XI. BUBONIC PLAGUE

**Definition.**—Bubonic plague (black death) is an acute infectious disease caused by the *B. pestis*, characterized by buboes, in the ordinary form, by subcuticular hemorrhages and an extremely high mortality.

The disease has existed in the Orient since its disappearance from Europe in the seventeenth century, and frequently breaks out as an epidemic. Since the outbreak in Hong Kong in 1894, it has spread to many civilized countries, but has obtained no firm foothold. In India it is still widely prevalent and very fatal, and has ravaged Manchuria during the present year.

The disease is caused by the *B. pestis*, transmitted to man by the rat flea. In California the ground squirrel serves as the medium of transmission also, and in Manchuria a species of marmot. These animals harbor a flea capable of transmitting the disease. Ordinarily propagated by skin infection, the pneumonic form, recently prevalent in Manchuria, is presumably contracted by inhalation. The disease has obtained a firm hold in recent years only amongst those peoples living in disregard of modern sanitary requirements, in respect to the presence of rats and fleas, and the opportunities given the fleas, as amongst barefooted races, to spread the infection.

**Symptoms.**—The period of incubation is stated at from two to five days. The onset is sudden and severe, with all the usual symptoms of a grave infection, the mental depression and anxiety and the prostration being especially marked. Pain in the lymph nodes affected, hemoptysis and hematemesis, injected conjunctivae, and hurried respiration are especial features of the disease.

Commonly recognized varieties of plague are:

(a) **PESTIS MINOR.**—Pestis minor, or ambulatory plague, with mild symptoms often not preventing the patient from remaining up and about, is rarely fatal. The buboes may suppurate, and the patient then becomes dangerous to the community because of the facility with which he may spread the infection from the unhealed ulcers.

(b) **ORDINARY FORM.**—This has been already described in part. On the third or fourth day the fever commonly remits, to rise again as the buboes suppurate—secondary fever—with dry tongue, delirium, stupor, collapse, and frequently death. The buboes may undergo resolution, or gangrene may result, but suppuration occurs in perhaps half the cases. Splenic enlargement and petechiae, often with bleeding from the mucous membranes, lungs, stomach and intestines, and extensive carbuncles, are present. In favorable cases the bubo suppurates and the symptoms begin to abate, the fever declining by lysis. In severe cases death results about the third, fourth, or fifth day from exhaustion, often with coma and convulsions.

(c) **SEPTICEMIC FORM.**—In this variety no bubo is formed, the disease being septicemic from the start, corresponding to the septicemic types of typhoidal and pneumococcic infection. The course is a rapid and severe one, often with extensive hemorrhage and ending in death in one to three days.

(d) **PNEUMONIC PLAGUE.**—This is the type recently so prevalent and so fearfully fatal in Manchuria. It is a pneumonia with the usual physical signs, often of the bronchopneumonic type rather than lobar, of great intensity, and due to the specific organism of the plague. Bright red, bloody sputum, with many plague bacilli, extreme dyspnea and cyanosis, coincident pleurisy and death within five or six days are further characteristics of pneumonic plague. A secondary type of lung involvement is described, presumably arising from septic emboli or aspiration of secretions from the upper air passages.

(e) **INTESTINAL FORM.**—This form is less common, and is characterized by intestinal pain and vomiting and bloody diarrhea, without buboes.

**Diagnosis.**—The diagnosis is established in solitary cases by find-

ing the bacilli in the pus from the buboes, in the blood culture or the sputum. In epidemics, the clinical features of the disease are sufficient for diagnosis, in all but the ambulant form. The agglutinative reaction occurs so late in the disease as to be of little value in diagnosis.

**Differential Diagnosis.**—This involves differentiation from acute septicemic diseases, from pneumonia and from diseases presenting glandular enlargement. There can ordinarily be no difficulty if the possibility of the plague be considered, and proper bacteriological measures be adopted. With the beginning of the plague in San Francisco, there occurred in Colorado an outbreak of multiple abscesses, many affecting the axilla and groin, and about a dozen cases were reported to the State Board. At the same time two Chinamen, believed to have come recently from the infected district, were reported as having buboes and sinuses in the left groin. The former were found to be mild streptococcic pyemia, and the latter two were the result of necrosis of the inner surface of the left iliac bone, also of streptococcic infection.

**Prognosis.**—This depends upon the type of the disease. Ambulatory cases commonly recover, while more virulent forms of plague have a mortality rate of thirty per cent. to nearly one hundred per cent.

## XII. ANTHRAX

**Definition.**—Anthrax (malignant pustule) is an acute infectious disease caused by the bacillus anthracis, occurring especially in cattle and among sheep, and occasionally, through accidental infection, in man.

Individuals who contract the disease are especially those coming in contact with animals, particularly sheep and cattle, affected by anthrax. The disease is the most widely spread infectious disease of the vertebrates, and many opportunities for infection offer, whether the live animals, or the skin, hair, wool, or other products be the source of the infection. Those sorting wool so frequently have this disease that it is known in England as "wool-sorter's disease." On the Continent it is spoken of as "rag-picker's disease." Those hand-

ling hides have furnished most of the cases in America, so far as I know. Infection from man to man is possible but not very frequent. The pulmonary and intestinal forms of anthrax presumably depend upon the inhalation or swallowing of the infected material. It is not established that insects convey the disease to man.

**External Anthrax.**—(a) **MALIGNANT PUSTULE.**—This occurs at some point most commonly not covered with clothing, where the skin may readily be abraded and infected, as upon the right side of the neck in porters carrying dried hides upon the right shoulder, as in numerous reported instances.

*Symptoms.*—Three days or so after the infection the abrasion becomes reddened, a papule develops, and shortly a vesicle containing bloody serum. Sharp burning pain may be present. The vesicle breaks, leaving a black scab in the midst of a reddened, inflamed area, with reddened lymphatic vessels extending outward around it. Secondary vesicles appear during the next day. Fever, prostration, sweating, vomiting, enlargement of the spleen, delirium, coma, collapse and death may supervene, the fatal result occurring in from three to six days from the first symptom. In many cases the constitutional effects are comparatively slight, pus forms, the eschar separates, and convalescence ensues.

(b) **MALIGNANT EDEMA.**—The infection here shows no external pustule, but instead a rapid and severe spreading edema, especially about the face, neck, or hands, accompanied with symptoms of general infection. Extensive sloughing may occur. It is notable that the patient often shows no sign of appreciation of the gravity of his condition.

**Internal Anthrax.**—(a) **WOOL-SORTER'S DISEASE.**—Those sorting wool in carpet factories before ventilating tables were used were especially subject to the pulmonary form of anthrax from inhalation of infectious dust. Sudden onset, fever, headache, shivering, obscure pains in the chest, and a feeling of utter prostration are said to occur. Sudden collapse and death are frequent. Cough, perhaps without expectoration, with dyspnea and oppression within the chest are common. The pulse is weak, irregular, and rapid.

(b) **INTESTINAL ANTHRAX.**—This rarely occurs in man, and is

thought to arise from swallowing infected material, generally as meat or infected milk. In addition to the constitutional symptoms mentioned, vomiting, pain and diarrhea are noted, with little fever. Prostration, convulsions, coma, collapse, and death may be expected in a few days.

Internal and external anthrax are not infrequently found in combination.

**Diagnosis.**—In the laboratory the *B. anthracis* may be demonstrated with especial ease in either the blood, or the pus or serum from the lesion. A moderate polynucleosis exists. Inoculation experiments are conclusive. The direct diagnosis may be made from the characteristic appearance of the pustule in many cases. In malignant edema microscopic assistance is necessary. The diagnosis in the internal forms may be suspected in those known to follow an occupation exposing them to infection, but can be made certain only by laboratory procedure. The differential diagnosis from various skin infections ultimately depends upon the same means.

**Prognosis.**—Malignant pustule usually recovers if treated promptly by surgical means. The other forms are commonly fatal.

### XIII. GLANDERS

**Definition.**—Glanders (farcy) is an equine disease caused by the *B. mallei* and occasionally transmitted to man. In the nose ulcerating nodules occur, and in the skin nodules which give rise to abscesses (farcy). Extensive lymphangitis and pyemic manifestations are features of the disease. Acute and chronic forms of both glanders and farcy are recognized. The disease in man commonly occurs in those exposed to accidental infection, as of a slight abrasion, with the organism present in the discharge from the ulcerating granulomata of the nose in a horse with chronic glanders, this form going unrecognized in many cases. Thus a knowledge that one comes in contact with horses is the first point in making the diagnosis of this rare disease.

**Symptoms.**—The incubation period is probably three to five days, the patient often suffering from malaise, nausea and anorexia during

the time. Swelling and redness with inflammation of the lymphatics at the point of infection are then noted, with enlargement of the neighboring lymph nodes. Should the infection have taken place in the nose the mucous membrane swells rapidly, with bloody and later purulent secretion. Ulcerations soon appear where the nodules in the mucous membrane have broken down. A pustular eruption develops and has been mistaken for that of small-pox. It is found especially upon the face and the larger joints of the extremities. Cough, purulent expectoration, arthritis, petechiae and submucous hemorrhage, sweating, diarrhea, convulsions, coma and death may follow within a few days. The chronic form has none of the severe manifestations noted in the acute type, but is recognized by the persistent nasal discharge, with chronic ulceration of the nasal mucosa.

In acute farcy, the infection is in the skin and lymphatics, from a local infection elsewhere than in the nose. The swollen lymph nodes, "farcy buds," suppurate and give rise to abscesses, pyemic symptoms supervene, and death occurs in ten or fifteen days. The chronic form of farcy develops much less quickly and severely, with little involvement of the lymphatics. Chronic ulcers remain after the "buds" have broken down.

**Diagnosis.**—This must generally depend upon laboratory methods and animal inoculation. Chronic ulceration in the nose, especially in stable men, etc., should suggest the disease, and occasionally a direct history of exposure renders the diagnosis fairly certain.

**Prognosis.**—Recovery scarcely occurs excepting in chronic farcy, and then in but fifty per cent. of the cases.

#### XIV. LEPROSY

**Definition.**—This is a chronic infectious disease, caused by the *Bacillus lepræ*, and occurring in a tuberculous form, an anesthetic form, and as a mixture of the two.

**Etiology.**—Neither race, age, sex, diet, climate nor elevation above the sea seem to have the slightest influence as to the occurrence of leprosy, although it develops more easily in those with poor tissue



resistance, associated with any of the above conditions, or otherwise, and, in general, in natives rather than in foreigners. The present view is against its hereditary transmission.

The bacillus is found abundantly in sections of the infected tissues. It is probable that it enters the body through the skin or through the mucous membranes of the upper respiratory tract. The disease rarely spreads in civilized communities to any appreciable extent, since the measures of isolation commonly adopted suffice to limit it. In leprosy colonies attendants occasionally contract it, and

in Molokai the frequency of infection reached nearly 10 per cent.

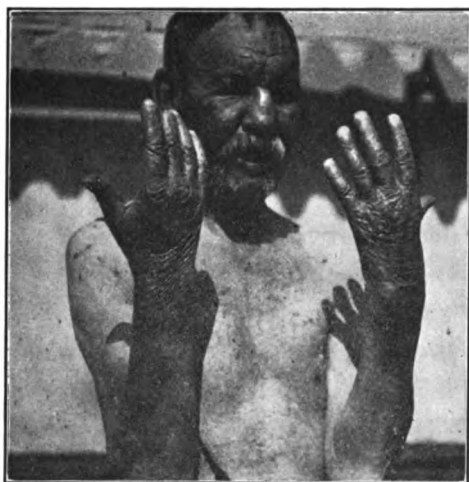


FIG. 30.—LEPROSY, ANESTHETIC TYPE. (Hospital for Contagious Diseases, Denver.)

**Symptoms.**—The period of incubation may be as long as several years. The symptoms depend upon the development of the bacilli in the skin and mucous membranes, in the surrounding lymph nodes, and around the nerve sheaths, with secondary infection as the lesions progress. As prodromal symptoms there may be irregular fever,

chilliness, loss of appetite, debility, and mental dulness. The tuberculous form is marked by patches of erythema, often hyperesthetic, most frequent upon the face, arms, knees, outer surfaces of the thighs, hands, and feet, and later by the development of macules, nodules, and pigmentation of the skin. Complete absence of pigment may be noted. The nodules run together and later break down, leaving chronic ulcers, with thick, indurated, scaly skin around, and great deformity from the cicatricial contraction. The spread of the process in the upper air tract leads to extensive destructive lesions of the mucous membrane and the deeper tissues of the nose, mouth, pharynx,

and larynx, with ozena, cough, dyspnea, aphonia, dysphagia, etc. The hair of the scalp is commonly retained, but that of other regions falls. The extensive thickening and tubercle formation, most marked in the face, give rise to the facies leontina of leprosy. Vision is lost in a majority of cases by the extension to the tissues of the eye. Death eventually occurs from exhaustion, from stenosis of the upper respiratory tract, or from deglutition pneumonia, resulting from laryngeal or tracheal involvement. A terminal diarrhea is not uncommon.

**ANESTHETIC FORM.**—The symptoms of this variety result from involvement of the nerves by the destructive process. Pain, hyperesthesia, or anesthesia are amongst the early features, and the areas affected may become white and scaly. The superficial nerve trunks may be felt as indurated cords, a peripheral neuritic process due to infiltration with the lepra bacillus and its products of inflammation. Bullæ, pigmentation, hyperidrosis, muscular atrophy, contractures, destructive ulcerations, loss of the hair of the affected parts, eventual dryness and scaliness of the skin, and necrosis, especially of the fingers and toes, are further features of anesthetic leprosy. The process is a slow one, and may extend over a score of years. In many cases the two forms described occur together.

**Diagnosis.**—This may be suspected in those who have lived in regions where leprosy is known to exist, and it is especially suggestive to learn that one has been exposed to contact with lepers. The diagnosis in isolated cases eventually depends, in the early stages, upon the finding of the bacilli in the nodules, serum, pus, sputum, nasal discharges, etc. They have been found in the blood. After extensive destruction of tissue occurs the diagnosis may be made without the finding of the infective agent in many cases, due regard being had to the possibility of the presence of syphilis, syringomyelia, and lupus. In all of these diseases similar destructive lesions may exist, but with especial features characteristic of each. In general, the most repulsive lesions pertain to leprosy.

**Prognosis.**—The disease eventually causes death, but its course may extend over half a lifetime. The average case terminates in death in about four years.

## XV. DYSENTERY

**Definition.**—The term dysentery signifies a disease characterized clinically by tormina and frequent stools containing blood and mucus, and pathologically by redness, swelling, and ulceration of the mucous membrane of the large intestine. The pathological term, colitis, fairly coincides with the clinical term dysentery. The different clinical varieties do not strictly belong together, and we shall discuss under separate headings the three different varieties, bacillary, amebic, and membranous. The chronic form will be considered in connection with the acute form with which it may occur.

## BACILLARY DYSENTERY

This is an acute infectious epidemic disease caused by the *Bacillus dysenteriae* of Shiga. It occurs also in the sporadic form, often called acute catarrhal dysentery. Further investigations have shown that there are at least four additional sub-varieties of the *Bacillus dysenteriae*, none of them being as virulent as the original. They may all cause dysentery, the cases being indistinguishable clinically. The bacilli have, however, well marked cultural, and especially agglutinative differences. The study has been a particularly difficult one, since many associated pathogenic organisms are thought by good authorities to have part in the causation of the intestinal lesions. The matter is still further complicated by the finding of one variety in the summer diarrhea of infancy. For the description of the different types of organisms the reader is referred to the special works.

**Etiology.**—The *Bacillus dysenteriae* is found only in the human intestine and its contents. The disease is transmitted by infection through the stools, the water supply being doubtless far the most common medium. Contamination of food supply through soiled fingers, direct transmission through handling soiled linen, and similar means play a part. The practice of fertilizing vegetable gardens with so-called "night soil" is responsible for its dissemination in the far East.

**Symptoms.**—The period of incubation is short, often not exceed-

ing 48 hours. Diarrhea with griping pain sets in suddenly, and as soon as the intestine is emptied of the major part of its contents, bloody and mucous stools appear, increasing in frequency until ten and even twenty stools are passed daily. Tenesmus is severe, and finally there is a constant burning in the rectum with almost constant desire to defecate. Loss of appetite, thirst, increasing fever, rapid pulse, weakness, and vertigo follow. In the severest cases 100 stools may be evacuated in 24 hours and delirium and death may be noted by the fourth day.

In the average sporadic case, as seen in temperate climates, no such severe course is expected. There were three deaths, all in babies under two years of age, in 98 cases occurring in Colorado, reported by me in 1893, clinically of this type, but reported before we knew anything of the bacteriology of dysentery. In these babies the disease would probably be classified now as enterocolitis rather than as dysentery. In the Southern States the disease is of fairly severe type and frequently becomes chronic. This course rarely occurs in the North. The severe dysentery of the Civil War, which incapacitated more men than any other single cause, was presumably of the type we discuss, since the liver abscesses of the amebic form were conspicuously absent. In the mildest cases the patient may be convalescing in three to ten days. Three or four weeks is a common duration in the South. The shading off into chronic dysentery in many cases in the tropics is a notable feature.

**Complications.**—Delirium is not infrequent in severe cases, often preceding death. In the tropics malaria is a frequent complication, and typhoid was a common one in the Civil War. Septic arthritis is not uncommon. The occurrence of liver abscess at once raises a presumption against bacillary dysentery. Albuminuria, pleurisy, and pericarditis may be noted. In the long continued cases, especially in chronic form, anemia and eventually edema of the legs and feet are very common.

**Chronic Form.**—In this acute ulcerations fail to heal, and chronic ulcers, especially at the colonic flexures, are found at the post mortem, extending down to the muscular layer, but ordinarily not undermined as in amebic dysentery. Thickening of the bowel wall

and such cicatricial tissue formation as to produce partial obstruction may be found. The ulcerative process occasionally extends up into the small bowel.

The acute febrile and catarrhal symptoms subside, and the stools consist of more or less normal fecal matter, with pus, blood, and mucus attached rather than intimately intermixed. The *Bacillus dysenteriae* may not be found. A long course, many months at least, characterizes the chronic form of dysentery. Some of the soldiers of the Civil War still have frequent relapses after fifty years have passed, it being evident from the appearance of blood, pus, and mucus in the stools that the ulcerations have never permanently healed.

**Diagnosis.**—The diagnosis of dysentery is easily made from the bloody and mucous stools and the tormina and tenesmus. The diagnosis of the bacillary type must depend upon the laboratory procedures, and especially the use of the agglutinative reactions, which naturally vary according to the strain of the *Bacillus dysenteriae* present in the given case.

**Prognosis.**—This is excellent in the sporadic dysentery of the North. In armies, and especially in the tropics, the death rate often exceeds 25 per cent. and may greatly exceed that rate. Shiga has shown that the mortality goes hand in hand with the upward extension of the disease from the rectum and sigmoid flexure. In Japan the average mortality is 20 per cent. or 25 per cent. The prolonged digestive disturbances following the disease and the liability to chronic dysentery which may cause trouble for life should be considered. The mortality is reduced to one-third the above figures under the recently introduced serum treatment. It is likely that the use of a polyvalent serum active against all the different strains of the bacillus of dysentery may still further reduce the death rate.

## XVI. GONORRHEAL INFECTIONS

We shall not consider the usual manifestations of gonococcal infection, but refer the reader to the special works. We shall consider certain features of the infection under the following heads:

(a) gonorrheal arthritis; (b) gonorrheal endocarditis; (c) gonococemia.

(a) **Gonorrheal Arthritis.**—This is an acute septic involvement of the synovial membrane, commonly of the periarticular tissues, and often of the tendon sheaths, due to the gonococcus or to a mixed infection associated with it. It is said to occur in twenty-two per cent. of all cases of gonorrhea (Gatther), although certainly not recognized in any such proportion. It is more common in males. The joint involvement may occur in gonorrheal ophthalmia. The gonococcus is the prime cause but the streptococcus and the staphylococcus, commonly associated with this organism, may take part in the arthritis.

The joints most frequently involved are the knee and ankle, probably then the wrist, but practically any joint may be involved. The sternoclavicular joint, that of the jaw, and those of the spine are often affected. One or two joints are more commonly involved, and generally not symmetrically. A knee and ankle upon one side constitute a very common form of involvement.

The arthritis usually develops during the height of the urethritis, often temporarily suspending the discharge, but it may equally well occur during a more chronic stage. The affected joint is swollen, reddened, commonly partially flexed, and extremely tender. The knee pan often floats. The inflammation remains long in one joint, not changing about as in acute rheumatism. It attacks the periarticular tissues as well, and the sheaths of the tendons attached to bones making up the joint are often involved. Marked muscular wasting occurs in the prolonged cases, and anemia and debility develop. The milder types are thought to be of toxic origin, while in the severe ones the finding of purulent or seropurulent fluid, with demonstration of the gonococcus or its associates, is common.

The disease tends to become chronic and may last for years, even, and without disorganization of the joint. Ankylosis and partial dislocation with disablement are frequent sequelae. Relapse is notoriously common. Leukocytosis is often marked. Endocarditis, pericarditis and iritis may develop, and gonococci may be cultivated from the blood in the former condition.

**DIAGNOSIS.**—This depends upon the demonstration of the causa-

tive organism in the urethral or vaginal discharge, or that from the eye, or upon the recently introduced complement fixation test. The non-symmetrical involvement and slow tedious course are suggestive. I have seen gonorrheal arthritis in the female when the vaginal and urethral canals were free from infection and only upon obtaining the secretion from the interior of the uterus was it possible to demonstrate the gonococcus.

(b) **Gonorrheal Endocarditis.**—This occurs in gonorrheal sepsis and may be of a malignant type. The organism has been cultivated from the blood and found upon the heart valves, the infection reaching the valves through the blood stream. Pericarditis and myocarditis develop. It is generally associated with gonorrheal arthritis, but not necessarily so.

(c) **Gonococcemia.**—This is a sepsis due to the gonococcus, ordinarily originating in the secondary suppurative processes beyond the urethral mucous membrane, and capable of exact diagnosis only in the laboratory. The symptoms are those of a virulent septicemia. Powers has reported a case of loss of an arm through acute septic involvement.

**Prognosis.**—Gonorrheal arthritis tends to recover after a long course. Gonorrheal endocarditis and gonorrheal sepsis are extremely grave.

## XVII. SEPSIS

**Definition.**—Under this heading we shall include toxemia, septicemia, and pyemia, all of which depend upon the growth in the body of pathogenic micro-organisms.

**TOXEMIA.**—Toxemia, replacing the old term “sapremia” which signified a poisoning from absorption of toxins from the bacteria of putrefaction, signifies the presence in the blood of the products of growth (toxins) of pathogenic organisms and the pathological processes resulting from the action of these toxins. Tetanus, diphtheria, and local pyogenic infections result in a toxemia.

**SEPTICEMIA.**—Septicemia signifies the presence, not only of toxins in the blood, but of the pathogenic organisms which have made the toxin. The infecting agent is not confined to its original seat,

if one be present, but acts in all parts of the body, and the removal of the original seat of infection has little effect upon the general process. Bacteremia signifies simply the presence of bacteria in the blood. In cryptogenetic septicemia, the focus of origin is not recognized.

**PYEMIA.**—This includes all that septicemia signifies, with the addition of local suppurative foci, due to infection caused by the lodgment of infected emboli. The secondary foci are commonly abscesses, but may show themselves in such processes as those of malignant endocarditis. The term septicopyemia adds little in significance to what we understand by pyemia.

**Etiology.**—The infection with one of the varieties of pathogenic organisms is the essential feature in the etiology of septic disease. The natural resistance of the body varies greatly but is never completely protective. The pyogenic cocci, streptococci and staphylococci are the most frequent causative agents, while the organisms of gonorrhea, pneumonia, typhoid, and influenza, bacillus pyocyaneus, *B. coli*, *B. proteus*, and several of the organisms causing various infectious diseases which we have already considered, may be the active agents. Toxemia may also occur from absorption of toxins from the intestinal tract, ptomains from decomposition of milk products, spoiled meats, etc. (sepsis intestinalis).

**Modes of Entrance.**—Most septic infections are associated with direct entrance of the causative agent into the circulation through a recognized break of continuity, as from a wound, fissure, puerperal lesion, typhoid ulcer, tonsillar inflammation, gonorrheal inflammation, ulcer, bed-sore, etc. A sloughing sore may furnish the organism and the place for its entrance.

**Symptoms.**—(a) **TOXEMIA.**—Toxemia may arise without recognizable local infection. No sharp onset is present in many cases. Chill or chilliness, malaise, prostration, rapid pulse, nausea, vomiting, and diarrhea, leukocytosis and various nervous symptoms may be noted. Fever is a constant symptom, frequently rising to 105° F. In such diseases as tetanus, diphtheria, pneumonia and erysipelas, the symptoms of the toxemia are a part of the recognized symptomatology of the general disease, and are due to the absorption of the



toxins from the local process, without the presence of bacteria in the blood.

(b) SEPTICEMIA.—The symptoms are similar to those of toxemia, but more sharp and severe, though the onset may be gradual. Definite chill is common, with high fever, commonly remittent, and in the severer cases intermittent. Anorexia, prostration, sweating, and the general “typhoid state” are common. Special peculiarities are associated with the different types of infecting organisms, and mixed infection is extremely frequent.

Cryptogenetic septicemia differs only in our inability to find the source of origin of the sepsis, sometimes even in the autopsy room. The prostate, urethra, appendix, gall-bladder, middle ear, uterus, sinuses, etc., should be carefully investigated. The streptococcus is the most frequent agent of the infection. Many of the infections are terminal ones in the wasting chronic diseases.

(c) PYEMIA.—This is preceded by toxemia and septicemia, and differs only in the addition of the suppurative foci mentioned. Its onset is announced by chills and sweats, and a generally increased severity of the symptoms which have characterized the preceding processes. A streptococcic rash is often present. Local signs will commonly point to the seat of the original infection. The streptococcus and the staphylococcus are the common causative agents. The lodgment of emboli in the lung points to their derivation from some source drained by the general venous system, whence they reach the right side of the heart and the pulmonic arterial distribution, while the lodgment of the emboli in the portal vein or the liver suggests appendicitis or other suppurative process in the territory drained by that vein. In septic endocarditis the diffusion of emboli may be general. Lymphatic distribution of the infection is less common and less rapid. Phlebitis and arteritis may occur. Minute emboli probably pass through the lung capillaries and cause foci of suppuration in the area of the systemic circulation. Emboli may be transferred to the left side of the heart through a septum defect. In appendicitis the process by which an artery becomes occluded probably involves septic embolism in the portal system, infecting the liver, abscess of the liver breaking into the lung, consequent thrombosis and embo-

lism in the distribution of the pulmonary veins, carrying of infected emboli to the left heart, and distribution through the systemic circulation. I have known the popliteal artery blocked, with amputation and recovery, presumably after such a course.

In addition to the general symptoms of the septic process already considered, we have in pyemia embolic manifestation in various organs, according to the distribution of the emboli. Infarctions occur in the lung, liver, spleen, kidney, brain, skin, muscles, eyes, joints, etc. Injuries, even in mildly septic streptococcic sore throat, may result in the development of local infection. I have known a fracture to become the seat of such an abscess. The mild type of pyemia described under the title "multiple abscesses" may be associated with an epidemic of streptococcic infection of the throat. One of a group of such cases reported by me had thirteen abscesses opened and finally recovered.

**Diagnosis.**—This depends upon the recognition of a more or less continuous fever, with the addition of repeated chills in pyemia, and the association in most cases with some septic wound, recent abortion, confinement, tonsillitis, gonorrhea, typhoid, or other process capable of giving rise to the infection. Leukocytosis and the finding of bacteremia are of great assistance. The slowly developing cases of sepsis are not commonly diagnosed for some days, while the acute sepsis following an autopsy wound may be recognized with the advent of the chill.

Absence of bacteria from the blood suggests a toxemia, but repeated examination should be made in case of doubt. Bacilluria may occur and point to infection of the kidney. Iodophilia is of some value as pointing towards sepsis.

The differential diagnosis between toxemia, septicemia, and pyemia has been sufficiently considered. Malaria is easily differentiated by a blood examination. Typhoid commonly shows the features of the disease, the Widal reaction and absence of leukocytosis, although the latter may be present in typhoidal sepsis. Endocarditis may commonly be recognized by the local cardiac features if they be sought for with care and skill. Minute emboli, giving rise to reddish-blue spots in the skin, may be found in certain cases

not definitely septic, but are more characteristic of septic endocarditis.

The more or less definite chills, the fever, prostration, and gradual failure seen in several diseases may suggest pyemia, but investigation shows some of the features upon which we may base a diagnosis. We refer to tuberculosis of the lungs, or, more particularly, of the kidneys, influenza, pyelitis, gall-stone disease, abscess of the liver, cancer of the liver, and blood diseases.

**Prognosis.**—A patient suffering with toxemia often recovers if the cause can be removed. Septicemia is extremely serious, varying somewhat according to the cause. Streptococcemia, pneumococcemia and pyemia are commonly fatal. The mild form of pyemia spoken of as “multiple abscesses” often recovers. The prognosis is always better if local abscess or other focus can be treated surgically. Some amelioration as to prognosis may be expected from the use of vaccine therapy. Relapse may occur after recovery seems assured.

### XVIII. CHOLERA

**Definition.**—This is an acute infectious disease, caused by the comma bacillus, indigenous in India, and occasionally epidemic elsewhere. It is characterized clinically by profuse watery diarrhea, vomiting, and collapse.

It is usually a waterborne disease. It is endemic in the delta of the Ganges River, being constantly propagated by the infection of the drinking supply through the impossibility of enforcing sanitary regulations. Thence it spreads periodically along the lines of travel, favored in its spread by warm weather, but not arrested by cold. Constant reinfection of the water supply occurs; uncooked food, infection from handling soiled linen, and from the house fly are also active agents in its spread. The comma bacillus is found constantly in the stools.

**Symptoms.**—The period of incubation varies from twelve hours to four or five days, generally two or three days. Dunbar distinguishes five forms of the disease, as follows:

(a) CHOLERA INFECTION WITHOUT DEFINITE SYMPTOMS.—The

patients are cholera carriers, and dangerous only to others in most instances.

(b) **CHOLERA DIARRHEA.**—This is the usual preliminary to a severe attack but may be recovered from in a few days. The stools are loose, yellowish, and frequent, and accompanied with colic and flatulence. Thirst, nausea, and languor may be noted. The patient must be isolated and the usual precautions taken until recovery occurs or the stools are proved non-infectious.

(c) **CHOLERINE.**—This is a mild type of cholera, presenting none of the serious symptoms to be described, with stools feculent instead of watery, and often recovering in 7 to 10 days.

(d) **PRONOUNCED CHOLERA.**—There are, following the preliminary diarrhea, frequent rice-water stools, nausea, vomiting, abdominal distress, muscular cramps, hiccough, and extreme prostration. Although the surface of the body is cold the rectal temperature is elevated to 101° to 105° F. Apathy and delirium are frequent, with cyanosis, pinched face, sunken eyes, rapid, feeble pulse, feeble voice, and cold skin. The urine contains albumin and abundant tube casts. The patient passes into the algid stage, with utter collapse, coma, and extreme cyanosis. The diarrhea ceases, and anuria results from the nephritis. Death occurs in most cases within a day or two. In the milder cases, the stage of reaction occurs, with disappearance of all the symptoms noted, and convalescence follows. Fatal relapse may occur.

(e) **COMATOSE STAGE.**—Somnolence and delirium appear, and finally coma. In extreme cases anuria and subnormal temperature supervene. Recovery results in many of the cases in which these two symptoms appear.

It should be noted that any stage of the disease may be absent. In cholera sicca the patient dies before the advent of the diarrhea. The stage of reaction may end in cholera-typhoid instead of prompt recovery. This occurs in about one-fourth of the severe cases, after the first three or four days of illness. It is regarded as a secondary septic infection, resembles severe typhoid in its symptoms, is often complicated by uremia, and commonly ends in delirium, coma, and death.

**Complications.**—Purpuric, macular, and erythematous rashes frequently occur during the stage of reaction. Acute nephritis has been noted. Diphtheroid colitis, suppurative parotitis, pneumonia, pleurisy, abscess, gangrene, thrombosis, etc., may occur. Recovery is often slow and complicated by gastro-intestinal disturbances.

**Diagnosis.**—Direct diagnosis is generally easy in a case of well-marked cholera occurring under conditions of possible infection.

**Differential Diagnosis.**—The finding of the comma bacillus excludes cholera nostras, although severe cases of the latter may so closely resemble cholera Asiatica as to be impossible of differentiation clinically. In case of doubt the bacteriological examination should be made promptly, the patient being isolated meanwhile.

The vomiting and purging from arsenical, antimonial, or mercurial poisons could scarcely be mistaken for cholera if the possibility of the occurrence be only remembered.

**Prognosis.**—The mortality is stated to vary from 20 per cent. to 80 per cent. Those lacking in resisting power from youth, age, or debilitating diseases commonly die. Even mild cases may require a guarded prognosis.

## XIX. TETANUS

**Definition.**—Tetanus (lock-jaw) is an acute infectious disease caused by the *B. tetani*, characterized by tonic spasms of the muscles due to the action of the toxins upon the nerve cells of the medulla and cord. An ascending neuritis extends centripetally from the point of infection.

**Etiology.**—The disease arises from infection by the causative organism of an external wound, although the exact site of infection is not recognized in certain cases. Punctured and lacerated wounds, and the unhealed site of the separation of the cord in the new-born are frequent sites of infection. In the West many cases, several of which have fallen under my observation, originate in injuries from barbed wire used in fencing. The danger of punctured wounds acquired in stables and barnyards has long been recognized, for the disease is frequent in horses, and the bacillus is found almost normally in horse manure and in garden soil. The incidence of the

disease in those working about stables is thus accounted for. Tetanus is most common in hot weather and especially in hot climates. The occurrence of the disease in lying-in hospitals, in general hospitals, and in the armies in almost epidemic form is practically a thing of the past. Limited outbreaks have occurred as the result of contamination of the wound after vaccination, but rarely from contamination of the virus. Diphtheria antitoxin was the proven medium of transmission in at least one endemic.

The great majority of cases follow wounds of the extremities. The disease may originate after the point of infection has become completely cicatrized, and the scar may reopen with the development of the affection. The cases occurring since the introduction of the blank cartridge and toy pistol are due to the carrying of the infection into the skin, where the conditions of the wound favor the development of the anaërobic bacillus. It is thought that the organism may be upon the skin in these cases, or in the coarse brown, earth-filled paper used as wadding, or in the salt-peter used in the powder, but no proof of any exact and constant method of the infection has been offered. I saw five cases of fatal tetanus in one week resulting from toy-pistol injury to the palm of the left hand, incurred in the act of loading a pebble into the barrel to serve as a missile. The opportunity for infection is obvious. The so-called idiopathic tetanus, so far as we now know, is a variety in which the point of infection has not been recognized.

**Symptoms.**—The period of incubation is one or two weeks in the acute, and more than two weeks, even two months, in the chronic form. In addition to these two forms a cephalic tetanus is recognized.

**ACUTE FORM.**—As prodromal symptoms there may be headache, anorexia, and slight fever, with stiffness of the muscles of the jaw and neck. Spasm of the masseters and other facial muscles appears (trismus), and the muscles of the mouth are often in persistent spasm (risus sardonicus). Severe general convulsive seizures follow, with tense abdomen, pain in the chest from spasm of the diaphragm, and opisthotonus, emprosthotonus, or pleurothotonus, according to the distribution and severity of the muscle spasm, the

first being the most frequent and characteristic. General rigidity (orthotonus) is common. During the spasms agonizing pain, especially in the chest, rupture of muscles, fracture of bones, noisy respiration from laryngeal involvement, cyanosis, and even death from asphyxia may result.

The spasms may be excited by a draft of air, noise, or movement of a limb by the nurse. Perspiration is often abundant. Profound exhaustion follows.

The pulse rises to from 120 to 140 per minute. The fever is generally moderate but hyperpyrexia (even to 110° F.) may occur. Respiration may be hurried at times, or mechanically suppressed during the convulsion. The reflexes are commonly increased. A persistence of the muscular contraction during the interval between the spasms is characteristic of tetanus. The mind is generally clear, but delirium is occasionally present. The general muscular spasm may prevent the introduction of an enema tube or a catheter. A moderate hyperleukocytosis is commonly present.

**CHRONIC FORM.**—Here all the symptoms are milder, intervals of relaxation of spasm occur, enabling the patient to take liquid foods, the intervals becoming longer and the spasmodic seizures less severe as the disease progress. Relapse may occur.

**CEPHALIC FORM.**—The form described by Rose depends upon infection in the distribution of the facial nerve, with spasm of the muscles involved in swallowing, occasionally of those of the neck and abdomen, and sometimes paralysis of the facial nerve.

**Diagnosis.**—A special feature is the history of a wound; and muscular stiffness, dysphagia, spasms, and the further symptoms described, suffice for the diagnosis in ordinary cases. The finding of the *B. tetani* in the secretion from the wound is decisive. No agglutinative blood test is available.

**Differential Diagnosis.**—**STRYCHNIN POISONING.**—This has been of interest especially in medicolegal cases. The history of ingestion of a poison, violent convulsions from the very onset of the trouble, complete relaxation in the intervals, the severity and brevity of the course, and the chemical or physiological detection of the poison should make discrimination certain.

**TETANY.**—The history of the disease, characteristic position of the fingers and toes, occasional laryngeal spasm, mildness of the course, presence of the phenomena described by Trousseau, and by Chvostek, and lack of general muscular spasm are characteristic.

In hydrophobia, the history of a bite, absence of lockjaw, and presence of spasm upon attempting to swallow; in torticollis the limitation and mildness of the symptoms; and in hysteria the general clinical picture should prevent error.

**Prognosis.**—Excepting as influenced by the serum treatment in selected and especially in early cases, the mortality in the acute form is 70 per cent. or 80 per cent., and practically 100 per cent. in the new-born. In the chronic cases and in the cephalic type the fatality is about 15 per cent. The longer the course the better the prognosis. Cases developing late after injury are favorable in outlook.

## B. FUNGUS INFECTIONS

### I. ACTINOMYCOSIS

**Definition.**—Actinomycosis (big jaw) is an infectious disease of cattle, occasionally transmitted to man, and caused by the streptothrix actinomyces, or ray fungus.

The disease occurs chiefly in the mouth and jaws, both in man and in animals, and the infection is carried by contact with the sharper portions of grass and straw, as the beards upon the head, upon which the ray fungus is normally found. Many cases have been noted in those accustomed to chewing grass or bits of straw. Those living upon farms or handling hay, grain, and cattle are the ones especially exposed to the disease. Most of the cases in America have been reported from the great stock-handling centers. Infection has occurred from water contaminated by the discharges of diseased animals. Bridge reports many cases of pulmonary infection from Los Angeles.

**Symptoms.**—A tumor with surrounding induration appears, usually upon the lower jaw or at the angle of the jaw, hard and brawny, and often mistaken for sarcoma. Eventually suppuration occurs,



a sinus opens either internally or externally, and the discharge is found to contain the yellow granules, visible to the naked eye, of the ray fungus. They should be proven microscopically. Burrowing, ulceration, and chronic course are common, perhaps ending in a chronic type of pyemia, as in one of my cases. Since the ray fungus is not at all certain to be found at a single examination a negative opinion in a suspicious case should be given only after repeated examinations. The abscess may contain gas with but little pus.

The intestinal infection most commonly involves the cecum and appendix, occasionally the sigmoid flexure. Metastases may occur, and the liver may be involved. The fungus may be found in the stools. The course is usually chronic, with septic phenomena. Pericecal abscess and peritonitis may be noted. About 20 per cent. of the cases of actinomycosis involve the abdominal organs.

The respiratory form may involve the larynx, and the lungs may be affected either primarily or secondarily. Both pathologically and clinically the disease offers a considerable resemblance to pulmonary tuberculosis, but there is a special tendency to the formation of fistulæ, to necrosis of ribs or other bones, and to chronic sepsis. Empyema may occur. The fungus may be detected in the sputum, at times with difficulty, because of its marked pleomorphism. The skin, tonsils, brain, and other tissues have been occasionally involved.

**Diagnosis.**—Although suspicion should be aroused by the finding of the signs mentioned, the proof of the diagnosis must come from the laboratory. The fungus may not be present in characteristic form, but as mycelial threads. The organism of mycetoma closely resembles it. The absence of tubercle bacilli in the sputum of chronic lung disease should lead us to the suspicion of actinomycosis.

**Prognosis.**—The local form often recovers completely with treatment. The chronic pulmonary and intestinal forms rarely recover.

## II. BLASTOMYCOSIS AND NOCARDIOSIS

In recent years a number of cases of lung infection have been reported by blastomyces, often secondary in cases of blastomycosis of the skin, and by streptothrix (nocardia). Infection by the latter

probably occurs through inhalation of infected dust. In Australia it has been noted after severe sand and dust storms. Both diseases bear the same general resemblance to pulmonary tuberculosis already noted in the case of actinomycosis. Cough, bronchitis, profuse expectoration, consolidation of lung, severe mixed infection (and in

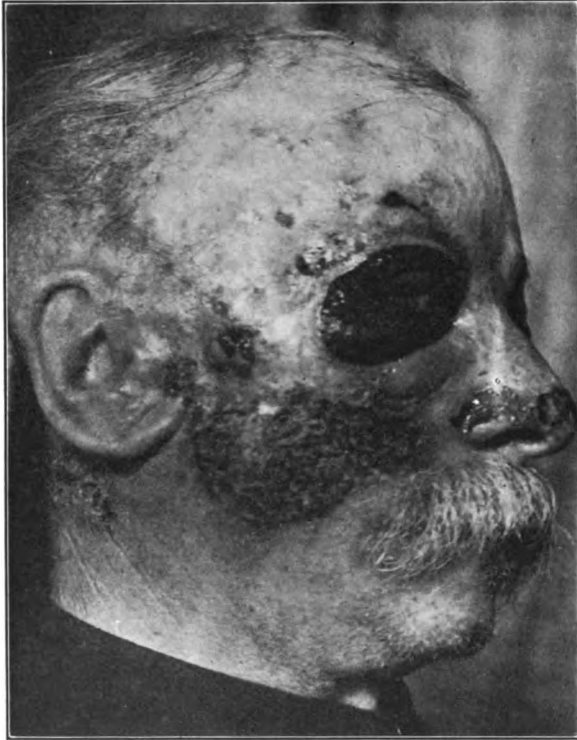


FIG. 31.—BLASTOMYCOSIS. (From the collection of Dr. A. J. Markley.)

streptothricosis, extensive cavity formation) with hectic fever, and a downward course are characteristic of these infections. They may be distinguished from tuberculosis, actinomycosis, and from each other only by the characteristic findings in the sputum.

**Prognosis.**—This is practically the same as that of pulmonary tuberculosis with secondary infection and extensive destruction of lung.

### III. ASPERGILLOSIS

The *aspergillus fumigatus* may cause a chronic disease of the lung with consolidation and cavity formation resembling the processes in pulmonary tuberculosis. Cattle and pigeons also suffer from the disease, the latter, at first especially, in the buccal form. Those coming in contact with cattle, pigeons, and grain, upon which latter the fungus grows, are especially subject to the disease. It has been especially noted in France. The diagnosis rests upon the finding of lung disease in a general way resembling tuberculosis, and the detection by cultural methods of the *aspergillus*. The prognosis is similar to that in the diseases mentioned.

## C. PROTOZOAN INFECTIONS

### I. MALARIA

**Definition.**—Malaria or malarial fever is an acute infectious disease caused by the *plasmodium malariae*, transmitted by the bite of infected mosquitoes and characterized by intermittent fever, or by a continued type of fever, and by splenic enlargement, with more or less anemia or actual cachexia.

The disease may be found in any region where the climatic conditions permit mosquitoes of the family *Anophelinae* to breed. It is still especially common and severe in tropical countries, but is becoming less prevalent in temperate regions with better cultivation of the soil, better drainage, and improved housing and sanitation. In temperate climates it is found only during the warm portion of the year, when the mosquito can carry the infection.

**Classification.**—Malarial fevers are commonly divided into classes, as follows:

(a) Tertian malaria, with an ague paroxysm every other day, caused by the *plasmodium vivax*. If the patient have a double infection a paroxysm occurs daily, the common type in the northern part of the United States.

(b) Quartan malaria, with a paroxysm every third day, or

oftener if more than one brood of parasites exists—two days out of every three, or daily. The parasite is the *plasmodium malariae*. The two parasites mentioned only rarely give rise to the pernicious forms of paroxysm.

(c) Estivo-autumnal malaria, with paroxysms daily or every second day, frequently overlapping because of their long duration, and then producing a more or less continued fever. Craig believes that there must be two varieties of the *plasmodium falciparum*, the quotidian, causing the daily paroxysm, and the tertian, causing the type corresponding in time to the usual tertian form, and responsible for probably 75 per cent. of the pernicious paroxysms of the disease.

(d) Malarial cachexia, a chronic cachectic condition depending upon the anemia and debility resulting from repeated malarial infection.

In order that the mosquitoes of the *Anopheles* family may become capable of carrying the infection, it is of course necessary that they have the opportunity to feed upon the infected blood of patients with malaria. Thus whole countries may harbor mosquitoes of the malarial-carrying family, yet show no malaria. For the life history of the various parasites the larger works should be consulted.

**Symptoms.**—Those of the first two varieties, constituting the group known as the regularly intermitting fevers, or agues proper, will be described together, since they vary but little excepting as to the time of the paroxysm.

The period of incubation varies from probably four or five days to fourteen or even twenty days and is probably not constant even for the same type of infection.

**THE PAROXYSM.**—This consists of the three stages, chill, fever and sweat, which together make up an ague fit. Headache, languor, yawning, occasionally nausea and vomiting, precede the advent of the chill. Then coldness and creepy chilliness develop into a sharp rigor, with shaking, chattering teeth and goose-flesh. The surface of the body is cold to the touch but the rectal temperature may rise to 105° F. or more. The pulse is small and rapid. The chill com-

monly lasts ten to fifteen minutes but in severe cases even an hour, this prolongation signifying increasing danger.

The cessation of the chill marks the beginning of the hot or febrile stage, with flushed face, congested eyes, bounding pulse, throbbing headache, and perhaps delirium. The fever remains at about the point reached during the chill, and the patient complains of thirst and of great heat. After half an hour or an hour, or even three or four hours, in severe cases, the fever falls by crisis. As it falls the patient passes into the sweating stage.

**SWEATING STAGE.**—Drops of perspiration appear upon the face and soon the entire body is drenched in sweat. The headache and discomfort disappear and the patient commonly falls asleep. Upon awakening he feels fairly well excepting for weakness. The duration of the entire paroxysm is from six to twelve hours, often much less in those long exposed to malaria. The different stages may be much modified as to character or length. Herpes labialis is frequent and the tender and enlarged spleen is commonly to be palpated during the paroxysm, subsiding somewhat after its close.

In the interval between the paroxysms the patient commonly resumes his occupation, and appears well except for the moderate weakness. If the chill recur daily, the patient must have a double tertian or a triple quartan infection; if every second day only, a single tertian; and if every third day only, a single quartan group of parasites. The mild cases recover without treatment, but relapse is extremely common, if the exposure continues.

**Estivo-autumnal Infection.**—This constitutes the severe and dangerous form of malaria with fever often irregularly remittent or continued in type, and not infrequently presenting the pernicious forms of infection to be described. It occurs chiefly in tropical and semitropical countries, and is common in the gulf region of the United States.

The period of incubation is thought to be about five days. The clinical picture is much less sharply defined than that of the intermittent types, since the paroxysms are longer and often overlap, producing a more or less continuous fever. The tendency of the paroxysms to "anticipate," the frequent absence of definite chill, the un-

certain and perhaps varying periods of development of the parasites, the frequent defervescence by lysis, and possible presence of multiple groups of parasites add to the confusion.

Prodromata, such as occur in the regular intermittent types are present. Then, with or without chill, the fever begins, rising and falling more gradually than in ague. Thus, with renewed infections, a remittent or almost continuous type of fever develops, and perhaps so devoid of paroxysmal rise or fall as to resemble typhoid. The temperature may remain constantly between 102° and 105° F. The spleen is enlarged, but other features of typhoid are wanting. The fever persists for a period varying from a week to a month, according to the type of infection and the efficiency of treatment. Slight jaundice may appear frequently, and deep discoloration not rarely, the so-called "bilious remittent fever" of older writers. Bronchitis is often noted.

**PERNICIOUS FORMS** ("Congestive Chills" of the Laity).—In addition to the mild form described, ending in recovery in a week, and the severer types with jaundice, vomiting and marked remissions and exacerbations (bilious remittent fevers) we may have in estivo-autumnal infections, especially in the tropics, pernicious forms as follows:

(a) *Algid Form*.—This comes on suddenly, with sweating and collapse, perhaps without chill or increased temperature. Anuria is common. The temperature finally rises, and perhaps becomes high, though its course is entirely irregular. Even in an imported case I have seen the patient pulseless in three hours, with every sign of impending death. The choleric form presents similar symptoms, but, in addition, vomiting and severe purging occur, dependent, it is believed, upon the blocking of the intestinal blood vessels by innumerable parasites.

(b) *Comatose Form*.—The attack begins abruptly with intense headache, followed by delirium, stupor and profound coma. Contracted pupils and stertorous respiration are frequently noted. Recovery may occur in twelve to twenty-four hours under vigorous treatment, but the danger in the attacks is very great. I have, however, seen profound coma lasting 60 hours in spite of the most vigorous

treatment, followed by complete recovery. The cerebral blood vessels are blocked by the parasites during the paroxysm.

(c) *Hemorrhagic Form*.—This is rare in the northern part of the United States, but extremely common in the tropics and especially in parts of Africa. Hematuria, or frequently hemoglobinuria, is the most common manifestation of the hemorrhagic tendency (black water fever). It may possibly be due to a special parasite. No regular type of malarial paroxysm precedes the development of the hemoglobinuria, and it may even occur without definite chill or fever, especially in those who have had repeated attacks of malaria. Cutaneous ecchymoses and bleeding from the mucous membranes may be noted. The administration of quinin sometimes produces hematuria, but the evidence that black water fever is dependent upon the use of this drug is certainly insufficient up to the present time.

Various subvarieties, cardialgic, dysenteric and pneumonic forms, have been described.

**Malarial Cachexia**.—This is an anemia resulting from the destruction of the red blood corpuscles by repeated attacks of malaria, associated with great splenic enlargement (ague cake) and a lesser increase in the size of the liver, a muddy complexion, emaciation, pigmentation of the skin, irregular fever and not infrequently hemorrhage from the mucous membranes and from the stomach, or into the skin. The red cells may fall in number to 1,000,000 with corresponding hemoglobin content.

A definite dysenteric form of malaria is recognized by the surgeons in the Philippines, often greatly benefited by treatment by quinin.

**Latent Malaria**.—It has been shown by Craig that malarial parasites of different varieties may remain in the blood after an attack of malaria for an indefinite time without producing symptoms and in some of the cases no definite history of a precedent attack is obtainable. In the children of native Philipinos such latent malaria is common. Craig's discovery that patients dying of other diseases occasionally showed in the blood from the spleen pigmented leukocytes, and tertian and estivo-autumnal parasites in the red cells, goes

far to account for certain anomalous attacks of ague. The parasites were much less numerous than during acute attacks. Craig proves conclusively that the seat of the initial malarial infection is in the spleen. This strongly suggests a consideration of the advisability of splenic puncture for diagnosis in obscure cases suspected to be malarial in origin.

Thomson states that a leukocytosis, very variable during the 24 hours, is found for months after the apparent cure of the malaria.

**Complications.**—Various complications may be found in malaria but they are not sufficiently definite and frequent to demand special consideration.

**Diagnosis.**—In the laboratory the essential feature is the finding of the parasites in the blood, either in the fresh or the prepared specimens. It is best not to administer quinin unless urgently needed (especially for fear of a pernicious paroxysm) until the blood has been secured for examination, preferably at the time of the chill or just preceding it. Apart from the finding of the parasite, malarial leukopenia, an increase in the relative percentage of the large mononuclears, poikilocytosis, anemia, and the finding of blood pigment, either free in the blood plasma or in the polynuclear cells, may suggest the diagnosis. Albuminuria and even acute nephritis may be detected by the examination of the urine.

**Direct Diagnosis.**—This is usually easy in the frank types of malaria and is based upon the generally known exposure, regularity of the symptoms, enlargement of the spleen, curative effect of quinin, and finally upon the finding of the parasite.

**Differential Diagnosis.**—This would be much simplified if the physician would keep before his mind the common causes of intermittent fever, aside from malaria. It is humiliating to acknowledge the frequency with which pulmonary tuberculosis in an easily recognized stage, pyemia, malignant endocarditis, pyelitis, syphilis and especially cholelithiasis are treated as malaria for an indefinite time before the diagnosis is corrected. It is not that these conditions are difficult of diagnosis, but that intermittent fever suggests malaria so irresistibly to certain minds that no differentiation is attempted or even thought of. Careful physical examination with investigation



of the blood and urine is sufficient in almost all cases to establish the diagnosis, and even the test by giving quinin is commonly sufficient when laboratory tests are unavailable. Widal reaction, examination of sputum and urine, investigation as to leukocytosis, and the Wassermann reaction, tuberculin tests, etc., give additional means for differentiating the various maladies which might demand consideration.

There is danger, unless the multiform manifestations of malaria be kept in view, of overlooking the malarial basis of supraorbital neuralgia, intermittent attacks of urticaria, hematuria, so-called dumb ague. All intermittent affections should be scrutinized with great care, and in this region at least, many prove to be non-malarial.

**Prognosis.**—The ordinary intermittent fevers recover with treatment in almost every instance. The remittent types are much more severe, especially in the tropics, but in the United States the outlook is very good. Nearly one-fourth of the pernicious cases prove fatal. The prognosis may be much improved by treatment designed to prevent recurrences, with the danger of chronic cachexia and the greater one of pernicious paroxysms. The outlook in malarial cachexia is good, but it demands careful treatment and generally removal to a non-malarial locality.

## II. TRYPANOSOMIASIS

**Definition.**—This is an infectious disease caused by the trypanosoma *Gambiense*, acute in onset, but becoming chronic, being then known as sleeping-sickness. The East Indian and Philippine Surra, affecting horses and mules, the Nagana or tsetse-fly disease of Africa, and several other diseases of lower animals are caused by different varieties of trypanosomes.

**Exciting Cause.**—The minute wormlike animal parasite found in the blood of man in sleeping-sickness is of the order Flagellata and genus Protozoa. The parasite is transmitted from man to man by the tsetse-fly (*glossina palpalis*), for about 48 hours only after the fly has obtained infected blood from a patient suffering with the disease. The living parasites may be found for two or three days longer

in the stomach of the fly, but are incapable of transmitting the disease, having in some manner lost their virulence. Bruce believes that other varieties of the tsetse-fly may act as bearers of the disease.

Sleeping-sickness is found in Uganda and on the west coast of Africa, the flies being especially abundant along the lake shores. Because of the recent opening up of the interior the disease has been spread widely, and over 100,000 deaths have occurred from sleeping-sickness in Uganda alone.

**Symptoms.**—The infection may be entirely latent, as the forms found in many of the lower animals have long been known to be. The incubation period in monkeys is about 20 days, but in man it may be months or years before any especial symptoms are noted, unless the trypanosome fever has been observed at the outset. This is a more or less irregularly continuous fever lasting a week or more, accompanied oftentimes by a diffuse erythema, and constantly by the enlargement of the lymphatic glands and spleen. Edema of the feet may be present. During this time the trypanosomes are present in the blood and lymph glands.

After a period of months or years, even five years, alternating febrile attacks and apyrexia occur, the latter perhaps constant for months together. The patient becomes dull, apathetic, feeble, with shuffling gait, and finally lethargic, and the last stage of infection, "sleeping-sickness," is under way. At this time the parasites may be found in the spinal fluid in practically every case. Rapid, feeble pulse, anemia, slight, irregular fever, lymphocytosis, tremors and muscular twitchings, loss of control of the sphincters, coma, and death are further features. Death may be hastened by some complication, such as septic meningitis, dysentery, or especially pneumonia. The duration may be from several weeks to a year.

**Diagnosis.**—This would be impossible in most instances without a knowledge that the patient had been exposed to the infection, excepting through skilled laboratory investigation. Suspicion should be aroused when one known to have been in Africa presents an irregular fever, universal enlargement of the lymphatic glands, a lymphocytosis, and a beginning mental dulness. At this time the blood, spinal fluid, and a drop of serum obtained by aspiration of

the lymphatic glands should be examined for the trypanosomes. The blood withdrawn from a vein and injected into the white rat or the monkey will reproduce the parasites in the animal's blood in about a week.

**Prognosis.**—Bruce states that every case is fatal sooner or later, but the recent successful treatment of certain cases with atoxyl gives ground for a better prognosis under circumstances favorable for treatment.

### III. RELAPSING FEVER

**Definition.**—This disease is caused by the spirochetæ of Obermeyer, and is so named because of the characteristic relapse occurring usually on the fourth to the seventh day of the remission, with successive febrile and apyretic periods thereafter. No characteristic anatomical lesions have been discovered. It has never appeared in the United States in epidemic form, and has so decreased in prevalence in Ireland and England as to be of comparatively little importance. It is still prevalent in India. Its frequent association with typhus fever has been mentioned, and in Ireland it received the name "famine fever" because of its prevalence during the terrible famines of the middle of the last century. The same conditions of filth, overcrowding, and destitution associated with typhus are found with relapsing fever.

The spirochete is found in the blood only during the paroxysm, although highly refractile bodies thought to be spores may be seen in the interval. Tietin proved that the bedbug may be the medium of transmission in certain cases. Nuttall states that the usual agent is rather the *Pediculus capitis* or the *P. vestimenti*.

**Symptoms.**—After an incubation period generally of five to seven days (decidedly variable) the onset is abruptly announced by chills, malaise, headache, and sharp fever ( $104^{\circ}$ - $105^{\circ}$ ). Vomiting may occur and sweating and sudamina are common. Delirium, jaundice, and enlargement of the spleen and liver are often noted, with tenderness over these organs. After 3 to 5, even 7 to 10 days, defervescence occurs, often with sweating. Collapse may be noted.

The patient is practically well, excepting for weakness, during

the intermission. After five to seven days a relapse occurs, generally milder and shorter than the first attack, and even a second and third relapse may be seen, generally progressively lighter in character. Convalescence is slow in proportion to the severity and number of relapses. No especial complications are characteristic of relapsing fever, though pneumonia, rupture of the spleen, ophthalmia, and peripheral neuritis may occur. Pregnant women run even a greater risk than in typhoid.

**Diagnosis.**—This depends upon the finding of the spirilla, which fortunately is a very certain and comparatively easy procedure. This disease is so rare in America that the diagnosis would probably be made in the sick room only in case of known exposure. The relapse will lead to an examination of the blood, unless this has been previously carried out. A serum agglutinative test may lead to the diagnosis in the apyretic period, when spirilla cannot be found.

**Prognosis.**—The mortality is 2 per cent. or 4 per cent., practically confined to the old and feeble.

#### IV. SYPHILIS

**Definition.**—This is a chronic infectious disease caused by the *Treponema pallidum*. It may be either hereditary or acquired.

**Etiology.**—Acquired syphilis occurs as the result of inoculation of the specific organism into the body. Though commonly the infection occurs from the secretion of an open lesion of syphilis, the disease may be carried by the blood during the secondary period. It is called a venereal disease simply because something like 70 per cent. to 80 per cent. of infections are acquired in sexual intercourse, generally illicit.

Innocent syphilis (*syphilis insontium*) may be acquired in the marriage relation or by innumerable methods, of which kissing is the most common. This method of acquisition is said to be particularly common in Russia because of local customs. Medical men, nurses, and midwives frequently receive the infection upon the fingers. Tattooing, using an infected cup, pipe or mouth instrument, perverted sexual practices, nursing an infected infant, arm to arm

vaccination, and any other means by which the organism may enter the tissues should be mentioned. The healthy mother may acquire an immunity to the disease through the bearing of an infected child, without having shown a primary sore or any symptom of syphilis. (Colles' law.) A majority of such mothers give a positive Wassermann reaction. If the mother be infected before impregnation, or early in the pregnancy, she is likely to bear an infected baby, but the child may escape, and especially so if the infection be a late one. Practically all persons, so far as we know, are susceptible to syphilis, excepting those possessing immunity acquired through having had the disease, or, in case of the mother, acquired by bearing a syphilitic child. This immunity is commonly complete, but may be destroyed by the most recent methods of treatment, as has lately been demonstrated. Those born of syphilitic parents may possess some degree of immunity.

Hereditary syphilis more frequently comes from the father (sperm transmission) than from the mother. It is not necessary that the father show any signs of the disease, and on the other hand he may show abundant signs of the disease and yet possibly beget a healthy child. If the father be carefully treated, healthy children are the rule after three years. Without treatment the results are utterly uncertain. I once treated both father and mother after a twelfth successive miscarriage had occurred from syphilis, and the next pregnancy resulted in an apparently perfectly healthy boy who lived to grow up without symptoms.

**THE PRIMARY LESION.**—Two, three, or four weeks after the inoculation (very commonly about three weeks) a red papule forms at the site of the infection, shortly enlarging, breaking down at the centre, and, with the infiltration of the connective tissue with round cells, presenting an ulcer, roughly of the size of the fingernail, distinguished by the buttonlike induration at the base, and yet freely movable over the underlying tissues. A slow hyperplastic induration of the neighboring lymphatic glands begins during the first week or two. A sclerosis of the adventitia of the arterioles may be detected at this stage.

Upon the lip the chancre causes much swelling, and with the

associated adenitis presents a striking picture. The sore in this region, which will not heal for weeks and has associated notable enlargement of glands, is extremely likely to be chancre in those under middle age, cancer in those beyond. The recollection of such a rough-and-ready rule of diagnosis by the physician of little experience with the disease will at least cause him to investigate carefully for further evidence. In the urethra the sore may be so small as to pass almost unnoticed, unless the induration and adenitis be carefully sought. In the female, if the sore be well within the introitus, it is easily overlooked.

**SECONDARY LESIONS.**—In a period varying between six weeks and three months, the secondary symptoms indicating constitutional infection appear. Slight fever, oftentimes remittent or intermittent, and continuing for weeks or months, is characteristic. Headache, pharyngitis and alopecia, usually diffuse, but occasionally circumscribed, and occasionally a specific onychia, with temporary loss of nails, may be noted.

Skin lesions are constant. The syphiloderm may be macular, maculopapular, papular or pustular. The favorite distribution is over the trunk and the anterior surfaces of the arms and thighs. The forehead is often covered, more especially with the later papular rash (*corona veneris*). The spots vary much in size, abundance and in color, the latter varying from a faint reddish to a brilliant reddish-brown, and the lesions may run together if the rash be abundant. In the throat the rash gives rise to a pharyngitis, and to a coryza in the nose. The rash persists for several weeks and may reappear, often as a papular syphilid. Somewhat later than the macular rash there may be seen, in typical cases, a maculopapular or papular eruption, and not infrequently a pustular syphilid, looking so much like small-pox that, in times of epidemic, the patients are not infrequently admitted to the contagious hospitals. The symmetry as to location of the rash, the peculiar reddish brown or coppery color, the absence of itching or pain, and the polymorphous appearance of the rash are points in the diagnosis. The ignorant patient commonly does not know of the presence of the rash.

At the time of the appearance of the eruption mucous patches

appear within the mouth, small ulcerations of a superficial nature, covered with a whitish secretion, and especially common upon the lips, tongue and tonsils. A general reddish erythematous inflammation is present. Mucous patches may also appear upon the mucous membrane of the vagina and anus, and even in the thin moist skin between the toes. At the margin of the skin at the anal and vaginal openings condylomata appear. Leucomata, whitish patches upon the tongue, are common, especially in smokers. At this time a fairly marked secondary anemia is present, with a fall of the red cells to half the normal in the severer types (syphilitic cachexia).

Iritis, retinitis, choroiditis, and rarely labyrinthitis may be noted, the first being much the most frequent. A very characteristic feature is specific periostitis, coming on late in the secondary stage, and affecting especially the tibiae. Tenderness, swelling and edema may mark the severe forms, and the pain is notoriously worse at night. The nocturnal headache of syphilis may be due to a periostitis of the cranial bones. Nodes may be present upon the shins or the cranium. Abortion is common in pregnant women at this stage, the fetus being frequently dead.

The secondary stage commonly lasts through several months, but some of the manifestations may persist for nearly a year. The mucous patches are an extremely common source of the contagion of syphilis.

**TERTIARY STAGE.**—The time of appearance of the tertiary lesions varies very greatly, the early manifestations coming before the disappearance of the secondary lesions, and the later ones years afterward. The characteristic lesion of this stage is the gumma, consisting microscopically of an aggregation of lymphoid and plasma cells and leukocytes, which undergo fatty degeneration and softening. Complete absorption may occur, but ulceration and sloughing are extremely common. Gummata may develop in practically any tissue, and the ulcerative and necrotic changes to which they are subject may cause extensive destruction in skin, mucous membrane, cartilage, bone, or in the structure of the internal organs. The destructive process with cicatricial contraction following it may give rise to stricture of the rectum, of the urethra, stenosis of the larynx,

pharyngeal deformities, etc. Necrosis of the nasal bones is the basis of the deformities of the nose so commonly seen.

Rupia, dactylitis, orchitis, myositis and amyloid degeneration of internal organs are further manifestations of the tertiary stage of syphilis. The lesions of this stage are much more marked and persistent in neglected cases and those having but little resistance from any cause.

**Hereditary Syphilis.**—The infant commonly shows the lesions of inherited syphilis at birth, or more commonly within a few weeks thereafter. The absence of the primary lesion is a striking feature. The congenital indications of syphilis are the emaciation and lack of proper development, bullae (pemphigus) upon the hands and feet, fissures about the mouth and anus, snuffles, mucous patches, and enlargement of the liver and spleen. Less common are hemorrhages, especially at the navel, separation of the epiphyses of the long bones, pseudoparalysis and hyperostoses. "One-third of all children procreated of syphilitic parents are born dead, and of those born living 24 per cent. die within the first six months of life." (Anders).

**EARLY SYMPTOMS AFTER BIRTH.**—The baby may be normal at birth, and soon show "snuffles" and ulcerations about the tonsils, with necrosis of the nasal bones in the severe cases, and later a sunken nose. Otitis media and deafness may follow. Deaf-mutism may be due to this cause. An erythematous or papular eruption is common and appears often first upon the buttocks, spreading over the body in many cases. The muddy complexion and shrunken skin give the characteristic "old man" appearance. Mucous patches at this time are a frequent source of infection to wet nurses and others. Onychia, dactylitis, splenic enlargement, anemia, cachexia, restlessness and failure of proper development are features of the disease. Enlargement of the liver is less characteristic than that of the spleen and because of the relatively large size of the liver in babies, harder to demonstrate.

**LATER SYMPTOMS.**—The child under treatment may recover from all the symptoms and present for a time the appearance of health. Retardation of development is the most striking feature, and the growing child may appear several years younger than the normal



age. Asymmetry of the skull, transverse enlargement, bulging forehead, depression of the bridge of the nose, saber-shaped tibiae from periostitis, non-development of the testicles, and radiating cicatrices about the angles of the mouth are commonly seen.

Interstitial keratitis, labyrinthine disease, iritis, and notched teeth are common manifestations of hereditary syphilis. Hutchinson described the notches at the cutting edge of the teeth, best seen in the central incisors of the permanent set. The teeth are also narrower at the edge than at the base, as seen from the front. One or more teeth may be absent.

**Visceral Syphilis.**—For the manifestations of syphilis in the central nervous system the section on nervous diseases may be consulted.

**Syphilis of the Lungs.**—The white pneumonia of the fetus is characteristic of hereditary syphilis. The disease may show its presence in the lungs by the development of gummata, especially in the bronchi, and near the root of the lung. In one of my cases Dr. Robert Levy found an ulcer of gummatous origin at the beginning of the primary bronchus, visible with the laryngoscope, from which, over a period of a dozen years, profuse hemoptysis repeatedly occurred. More common and more characteristic is the chronic interstitial pneumonia, simulating fibroid phthisis, often associated with bronchiectasis, affecting the root of the lung chiefly, but not infrequently the pleural surface.

**SYMPTOMS.**—The disease assumes the form clinically of chronic bronchitis or chronic fibroid phthisis, accompanied by a long continued fever and by expectoration devoid of tubercle bacilli and of elastic tissue.

**DIAGNOSIS.**—In the event of amyloid disease or other visceral manifestation of syphilis the diagnosis may become very probable and great improvement under special treatment renders it practically certain. The Wassermann test is of great value in case of doubt.

**Syphilis of the Liver.**—Diffuse hepatitis is common in congenital syphilis. Gummata are of frequent occurrence, even as large as the fist, and causing most marked deformity of the liver, simulating the nodules of cancer. After absorption the cicatricial contraction may further distort the liver. The capsule of Glisson may be affected

and by thickening and contraction cause marked lobulation. Amyloid degeneration is common.

**SYMPTOMS.**—The liver is often enlarged and frequently very irregularly so. The left lobe more commonly shows the more or less indolent enlargement from gumma. The frequent mild jaundice, ascites and disturbances of digestion suggest cirrhosis of the liver. Long continued fever may be present. Enlargement of the spleen is associated. Pain and tenderness, especially over a gummatous nodule, and frequently associated with a perihepatitis, perhaps with audible friction, may occur. Marked change in the shape of the liver, due to degeneration and absorption of gummata, and the contraction of cicatricial tissue is not uncommon. Pressure upon the portal vein and its branches, due either to gummata or perihepatitis, may obstruct the venous current and cause ascites.

Amyloid disease causes great and uniform enlargement with thick, round, palpable, painless edge, in typical cases. Coincident enlargement of the spleen and albuminuria with tube casts, polyuria, anemia and perhaps dropsy may be noted.

**DIAGNOSIS.**—We must consider especially cancer of the liver and echinococcus disease, both of which give rise to irregular nodular enlargement. The history of the infection, positive Wassermann test, change in the shape and size of the liver under treatment, and evidence of syphilis elsewhere, commonly render the diagnosis possible.

**Syphilis of the Rectum.**—This is not rare, especially in women. Stricture is due to the development of gummata under the mucous surface, near the internal sphincter, and obstruction results. If the gummata ulcerate, pain is present, blood and pus are passed, and the cicatricial contraction results later in still greater obstruction.

**Other Forms of Visceral Syphilis.**—Gummata may develop in the esophagus, small intestine, heart wall, kidneys and testicles, but the symptoms are those of a growth rather than specifically those of a gumma. The diagnosis can be made only by a consideration of the history and other signs of syphilis. The development of signs and symptoms of marked cardiac disease in one with syphilis should lead to the suspicion that it is of syphilitic origin. Sudden death is said

to occur in one-third of the cases and the prognosis should be made accordingly.

The great frequency of syphilitic endocarditis and gummatous pericarditis, and frequent complete obstruction of the caliber of the arteries by specific endarteritis should be given due weight in the consideration of myocardial disease.

The characteristics of the urine in amyloid disease of the kidney have been discussed.

**Diagnosis of Syphilis.**—The history should be carefully obtained, giving due credit to all positive features. Denial of the infection should not weigh in the least against reasonably clear clinical evidence, since the patient may intentionally deceive, or may believe syphilis to be absolutely of genital origin, may be a poor observer, or too ignorant to be depended upon, or the manifestation of the infection may have been so slight that even the patient's medical attendant may have formed an incorrect opinion upon which the patient bases his statement. In case of doubt, and especially in the face of fairly suggestive evidence of syphilis, no negative opinion is justifiable. Fortunately time generally renders positive diagnosis possible, even when laboratory tests are not available.

In addition to the inquiry as to a primary sore and investigation for the sore or its scar, which should always be carried out, inquiry should be made as to rash, alopecia, glandular enlargement, mucous patches, sore-throat, soreness about the anus or vagina, headache, pain in the shins, abortions, diplopia, or paralysis of cerebral nerves. Examination should be made for adenitis, scars upon the soft palate, tibial irregularities due to periostitis, Hutchinson's teeth, keratitis, iritis, irregular or unequal pupils from the latter cause, Argyll-Robertson pupil, atrophy of the testicle, destruction of the nasal septum, rectal stricture, etc. In examining women, married ones especially, it is often necessary to be guided by signs and symptoms rather than to inquire too closely as to the history.

**LABORATORY DIAGNOSIS.**—As soon as the ulceration of the initial sore develops, skillful examination should demonstrate the presence of the spirochete. Fortunately we have the recently introduced Wassermann test, a serum reaction of the greatest depend-

ability when carried out by competent investigators. It is imperative that the test be made only by such, since the chances of error are very great. The positive reaction in the hands of a competent observer is to be regarded as absolute evidence of syphilis, and it is to be found in nearly every case of syphilis with any manifestation of the disease present, and in at least half of the apparently cured cases. The effect of mercurial treatment, of the giving of salvarsan and the use of alcohol in rendering the test negative must not be overlooked. The iodids apparently do not interfere with the test as judged by the most recent evidence upon this point. Justus' blood test and the Noguchi butyric acid test are of secondary importance.

**DIAGNOSIS OF HEREDITARY SYPHILIS.**—The history of repeated miscarriages without obvious cause is very suggestive, and a history of several babies dying at or shortly after birth almost equally so. Pemphigus, snuffles, mucous patches, scars about the mouth, the eye and ear signs, notched teeth, lack of proper development, and bony changes have already been considered. Even though children of syphilitic parents appear healthy, according to the so-called Profeta's law, the Wassermann test is found to be positive in a majority of them. They possess at best but a partial or temporary immunity as a rule.

**DIAGNOSIS OF VISCERAL SYPHILIS.**—This depends upon the history of the disease or of other clinical manifestations of syphilis, since the visceral symptoms are not dependent upon syphilis per se, but upon the arthritis, tumor formation, etc., caused by it. The Wassermann reaction is of the utmost value in case of doubt. The result of the tuberculin test may clear the diagnosis. Specific treatment has little influence upon certain late syphilitic affections (paresis, tabes), and the therapeutic test is therefore not dependable in them.

**Prognosis.**—This is good if only the treatment be efficient and long continued, and reasonable certainty of avoidance of transmission of the disease and of the occurrence of the late manifestations of syphilis may be obtained. No positive assurances are ever justifiable upon these points. The occasional malignancy of syphilis, especially in those broken down by alcoholic and other excesses, and

by disease, must be noted. I have seen death from cerebral syphilis in less than six months after the first sign of the initial sore. Since the acquisition of our possessions in the East, the malignancy of syphilis acquired from foreign races has been frequently commented upon, and I have seen several illustrative cases. The lack of resistance due to tropical residence should be given due weight in this matter.

## V. AMEBIC DYSENTERY

**Definition.**—Amebic or tropical dysentery is an acute infectious disease generally caused by the *entameba histolytica* (ameba dysenteriae), but, in the United States at least, also by the *E. tetragena*, with the symptoms already enumerated under the general description of dysentery. It is especially characterized by the tendency to give rise to abscess of the liver, and by chronicity. Craig has recently stated his belief in the identity of the two organisms mentioned.

**Etiology.**—It is a disease of tropical and semitropical countries, but sporadic cases occur at least as far north as the fortieth parallel in the United States. It is water-borne and may thus be contracted in the same manner as typhoid fever and cholera. The use of infected water as an enema seems the probable source in certain cases. Infected vegetables are a common cause.

**The Specific Organisms.**—The *entameba coli* is a common and harmless organism in human stools. The pathogenic amebae are found in great numbers in the stools, but especially in the shreds of mucus or pus, and in the expectorated pus from the liver abscess when it breaks through into the lung. The examination of the still-warm stool is desirable for the certain detection of the amebae, the movement of the ameboid processes of the ectoplasm being then distinguishable. Special training is necessary for the certain differentiation of the pathogenic organisms, and Strong emphasizes the statement that “the ameba must be found living and motile.” In the tropics or in very warm weather the microscopic slide need not be warmed for the examination. Care must be taken to exclude urine from the specimen to be examined, as it causes the cessation of movement of the organism.

The ulcerations caused by the ameba are chiefly found in the colon and tend to undermine at the edges, contrary to the findings in bacillary dysentery. Great thickening and very extensive ulcerations characterize the chronic cases. The lower portion of the ileum may be involved. Constriction of the caliber of the bowel is not uncommon from cicatricial contraction. The beginning lesion in the liver in case of abscess formation is essentially a local necrosis, the different patches running together to form the abscess in the severe cases.

**Symptoms.**—The incubation period is from seven to eleven days in kittens, according to Craig's report. It is likely that this represents approximately the time in man.

The onset of the acute form is like that of the bacillary dysentery already described in many cases, but may be milder, with a preliminary diarrhea lasting several days. In mild and well treated cases the appearance of blood and mucus in the stools may be lacking, recovery probably occurring before the development of any extensive ulceration. In the chronic form there are alternating periods of looseness and constipation until the disease is established. Moderate fever and rapid emaciation are noted. Sharp hemorrhage from the bowels occasionally takes place, and perforation may be the cause of death. A moderate leukocytosis is found. Sloughs may be passed in the stools. The acute form may cause death in the first ten days, but the tendency is toward recovery or toward a chronic course. In the latter the loose stools continue, with gradual loss of flesh and strength, anemia, prostration, dyspnea, palpitation and vertigo. The mind is commonly clear until death approaches.

**Complications.**—Suppurative arthritis seems to be less frequent than in the bacillary form, while hepatic abscess, perforation, hemorrhage and bronchopneumonia are more common.

**HEPATIC ABSCESS.**—This is the most serious and frequent complication of amebic dysentery, occurring in about 20 per cent. of the cases. Although most frequent during the acute period it may develop at any time, even after the dysentery has apparently ceased. About 70 per cent. of the cases show a location in the right lobe and in about half the cases the abscess is single. The rarity in the female

sex has been thought to be due to the less frequent addiction to alcohol, which undoubtedly predisposes to the complication. The amebae reach the liver through the portal vein, and probably also in some cases by migration through the intestinal wall and direct invasion of the liver from the free surface.

Futcher, Strong and Rogers, in different reports, found secondary bacterial infection in slightly over half the cases, most often by staphylococci, streptococci and colon bacilli. The material in the abscess is thick, grayish or brownish red, with fragments of necrotic liver tissue, and, under the microscope, swollen and fatty degenerated liver cells and fragments of cells, blood cells, fat globules and amebae. Polynuclear leukocytes are comparatively few in this type of abscess though present in great abundance in the ordinary bacterial abscess. The amebae may not be found in the contents of the center of the abscess but may be obtained in scrapings from the more active processes in the wall of the cavity, or in the expectorated pus in case of pulmonary involvement.

*Symptoms.*—Pain is a frequent symptom, generally dull and aching, and located either in the liver or in the region of the right scapula. Tenderness, with pain in the regions mentioned, may be found upon pressure. An irregular fever is commonly present, often resembling that of malaria, so that, with the chills and sweating, the diagnosis is confused in malarial regions. The pulse increases in frequency and may reach the height seen in typhoid fever. An increase in the polymorphonuclear leukocytes to 15,000 to 20,000 or more is commonly present. The skin is discolored in such a manner as to suggest the diagnosis to the experienced eye in many cases. It is sallow, pale or definitely jaundiced. Considerable loss of flesh is present as the case advances.

The liver becomes enlarged if the abscess be of any size, most often in the region of the right lobe, and may produce notable bulging. The superior border of the liver dulness may bulge upward sharply either in the front or back. Because of the frequent presence of the perihepatic inflammation a friction rub may be heard and even felt. A small abscess may not show itself in such a way as to be detected by physical examination. In that case the knowledge of

the dysentery, leukocytosis, fever and general symptoms may justify a provisional diagnosis of hepatic abscess.

Aspiration should be performed over the most prominent portion of the liver if the abscess be suspected. Because of the thickness of the contents the needle must be of fair size and the negative pressure good.

*Course.*—The abscess commonly ruptures into the right pleura or right lung, into the peritoneal cavity, or causes death by exhaustion if not operated upon. The hepatopulmonary abscess, with the less frequent hepatopleural invasion, is probably present in 2 per cent. to 7 per cent. of the cases of amebic dysentery. The invasion of the pleura and lung may occur without gross lesion of the diaphragm.

The abscess in the pleura may present the same symptoms as those of an ordinary empyema. In the lung it commonly perforates a bronchus and the pus is discharged by coughing, with the characteristic anchovy sauce expectoration. Rupture into the peritoneal cavity generally causes a fatal peritonitis. I have noted rupture into the stomach with vomiting of the pus, in one case, and a retrocecal abscess opening into the loin in another. Recovery may ensue after rupture into the bowel.

*Prognosis.*—Amebic abscess of the liver may recover by fortunate perforation or operation, but the mortality probably averages 70 per cent. It is even greater if the lung be involved.

**OTHER COMPLICATIONS.**—Perforation of the intestines occurs in much the same manner as in typhoid fever, and is generally fatal. As in enteric fever, however, adhesions in advance may permit of the formation of an abscess, generally around the cecum, with recovery. Intestinal hemorrhage of any serious degree is comparatively rare, the characteristic thrombosis of the intestinal vessels not infrequently leading to complete arterial occlusion which prevents such bleeding.

The appendix is occasionally involved in the inflammation, but this can rarely be determined, since the involvement of the cecum completely covers up the symptoms.

Sprue or psilosis, or at least an atrophic condition of the in-



testinal mucosa, with inanition, anemia, liquid stools and sore tongue, follows in some cases of prolonged amebic dysentery.

**Diagnosis.**—This must depend upon the finding of the amebæ in the stools, living and motile. Urine in the stools and exposure to cold stop the ameboid movements. Material for this examination may be obtained by the passage of the rectal tube. If an hepatic abscess be opened, scrapings from the wall should be obtained. The pus from the lung abscess always contains amebæ, though prolonged search may be required. The determination of the presence of a leukocytosis, absence of malarial organisms and of the Widal reaction may all be required in case of doubt as to diagnosis.

**Prognosis.**—The mortality is probably 5 per cent. in cases seen early, and treated with skill and persistence, but it is vastly greater in soldiers and others exposed to hardships. The tendency to relapse is notable. Many chronic invalids have returned to America from the Philippine Islands. In children the prognosis is stated to be better than in adults. Those cases not recovering inside of a year either die or linger on incurable.

## VI. MEMBRANOUS DYSENTERY

Membranous, pseudomembranous, or diphtheritic dysentery should be subdivided as follows:

(a) **Bacillary Type.**—This is a true bacillary dysentery with the formation of a yellowish false membrane instead of the ulcerative lesions described. The whole inner surface of the colon may be involved, but the tendency is toward membranous formation at the folds of the mucous membrane. This variety of bacillary dysentery is found chiefly in the tropics, and differs from the usual form in its greater severity, much higher death rate, and the passage of extensive portions of membrane from the bowel. Of the minority that recover, many suffer a protracted convalescence. In addition to the complications heretofore considered we should mention ulcerative endocarditis, nephritis and a late myocarditis. Perforation is especially to be feared.

(b) **Secondary Pseudomembranous Dysentery.**—This is a terminal

infection in chronic wasting diseases of many varieties, and is occasionally found in acute pneumonia. The essential features are a mild dysentery, and a false membrane, but the disease has no relationship to bacillary or amebic dysentery.

## VII. SPRUE

**Definition.**—Sprue (psilosis) is a chronic disease described by writers upon tropical medicine, probably of microbic origin, and having as a chief anatomical feature an atrophy of the coats of the bowel. Soreness in the mouth and rectum and a red tongue are noted. The stools are said to be irregular, soft, copious, and pale in color, often frothy. Loss of strength and cachexia are common. It follows after exhausting diseases, notably dysentery and diarrhea. The course is likely to be a chronic one, often with intermissions, and a tendency to relapse is pronounced. The cases are likely to die eventually from the long continued diarrhea and inanition.

## VIII. KALA AZAR

**Definition.**—Tropical cachexial fever or dum dum fever is a disease caused by a protozoan organism, the Leishman-Donovan body, epidemic in parts of India, and occasionally seen in other tropical parts of the eastern hemisphere, and even in Europe, in returning invalids. Europeans have rarely been subjects of the disease, but Petrone reports cases in Italian children differing from the usual type.

**The Parasite.**—This is found in the blood after staining with a stain of Romanowsky type, as a round or oval body, two to four microns in diameter, with a macronucleus and a micronucleus. The bodies have been found in the polymorphonuclear leukocytes in the circulating blood, but are especially to be sought in the liver and spleen, bone marrow, intestinal ulcers, mesenteric glands, etc.

The method of infection is possibly by the bedbug, but inoculation experiments have not succeeded in transmitting the disease. Even the exact status of the parasite biologically is not settled.

**Symptoms.**—The disease begins with headache, chill, vomiting, and fever, remittent and occasionally intermittent. The spleen enlarges markedly and the liver to a less extent. After two to six weeks of fever, an apyrexial period follows, and the fever and apyrexia may then alternate, with varying periods. Anemia, cachexia, emaciation, subcuticular hemorrhages, bleeding from the gums, diarrhea and dysentery, swollen joints, icterus, sweating and edema of the extremities follow.

The immense enlargement of the spleen, and the extreme secondary anemia with leukopenia are most characteristic features of the examination. The duration is from six months to more than a year.

**Diagnosis.**—This depends upon the demonstration of the causative organisms in the blood (found in 75 per cent. of the advanced cases), drawing it from the spleen or liver if necessary. The symptoms mentioned would fairly suffice for the diagnosis in the countries where the disease is endemic.

**Prognosis.**—The mortality is about 96 per cent. Rogers claims that this can be lowered to 75 per cent by the treatment with quinin.

## D. METAZOAN INFECTIONS

### I. DISTOMIASIS: FLUKE INFECTIONS

Stiles recognizes four types of infection in man by trematodes:

(a) "A pulmonary distomatosis, with cerebral or other infection as secondary";

(b) "An hepatic distomatosis, with splenic or intestinal infection as secondary";

(c) "An intestinal distomatosis"; and

(d) "A venal distomatosis." In addition we find recorded instances of

(e) "An ophthalmic distomatosis (very rare) which may be an accidental secondary form of hepatic distomatosis."

The life cycle has not been fully worked out in many of the flukes.

(a) **Pulmonary Distomatosis.**—The parasite is the *Paragonimus*

*westermanii*, a fluke 8 to 16 mm. long and half as broad. The brownish-yellow oval-shaped eggs are found in the sputum of the infected individual. They measure about .09 mm. by .06 mm.

The parasite is found in cysts in the lung, preferably the upper lobes, but also in various other organs. The disease has been chiefly studied in Japan, but cases often occur in China, Formosa, Corea, and, of more especial interest to Americans, in the Philippine Islands. A few cases have been reported in America. Some of the lower animals are infected in eastern countries. The exact method of infection is unknown.

**SYMPTOMS.**—These relate chiefly to the lungs, and the especial and characteristic symptom is hemoptysis. The blood is at first expectorated in a very small quantity, perhaps merely streaking the sputum. Later it may become rusty, and occasionally profuse arterial hemoptysis occurs. Increasing cough and expectoration, more marked after exertion, soon appear, with secondary anemia and marked weakness.

The sputum contains many eggs, with blood, pus and epithelial cells. The worm may be expelled with the sputum. In advanced cases signs similar to those of fibroid phthisis may be found. Symptoms derived from the location of the parasites in the brain, liver or other organs may be present.

**DIAGNOSIS.**—This depends upon the finding of the eggs in the sputum. In Japan the peculiar type of hemoptysis suffices for a reasonably certain diagnosis.

**PROGNOSIS.**—Many of the patients die of exhaustion. Death from hemorrhage seems to be much less frequent than in pulmonary tuberculosis.

(b) **Hepatic Distomatosis.**—The ordinary variety of liver fluke is the *Opisthorchis sinensis*, found in Japan and the Far East, but there are at least five other parasites causing symptoms practically identical. Some of the lower animals are subject to infection. The method and source of infection are unknown in most of the varieties. Hepatic distomatosis is found not only in the Orient but extensively in Siberia, occasionally in Europe, and a few cases have been reported in the United States.

**SYMPTOMS.**—In light infections there may be none; in severer ones, enlargement and tenderness of the liver, irregular diarrhea, perhaps bloody stools, pain, jaundice, ascites, slight fever, anemia and death from exhaustion are prominent features. The course of the disease may extend over several years.

**DIAGNOSIS.**—This depends upon the detection of the ova of the fluke in question in the stools. The presence of bloody diarrhea in one with an enlarged liver should suggest the examination.

**PROGNOSIS.**—The mortality in a large series of cases in Japan, reported by Katsurada, was 16 per cent.

(c) **Intestinal Distomatosis.**—This is due to the *Fasciolopsis buskii* and at least four other varieties of fluke. This disease is found only in Asia and Africa. The source of infection is unknown.

**SYMPTOMS.**—These are said to be indigestion and bloody diarrhea.

**DIAGNOSIS.**—This must depend upon the examination of the stools for the ova.

(d) **Venal Distomatosis (Bilharziosis).**—The parasite in the African disease is the *Schistosoma haematobium*, and in the Asiatic variety, the *Schistosoma japonicum*. It lives in the veins of the liver and deposits its eggs in various organs, the ova wandering frequently to the intestines and bladder, and being found in the blood clots voided with the stools and the urine. The source of infection is still unsettled.

**SYMPTOMS.**—The most striking feature in the urogenital infection is hematuria, commonly associated with pain in the back and pelvis, cystitis, vesical calculus and occasionally nephritis. Pain on micturition is frequent. Urinary fistulæ may be present. In the rectal infection diarrhea with bloody stools is noted, with local rectal distress. Anemia and debility develop and the patient is often carried off by some intercurrent infection.

**DIAGNOSIS.**—This depends upon the detection of the ova in the urine or stools, a thinly spread slide and low magnification being desirable. Eosinophilia may be detected early in the disease.

**PROGNOSIS.**—This is highly unfavorable, except in the mild cases.

## II. INFECTION BY NEMATODES

The diseases due to the round worm will be considered under the following headings:

- (a) Filariasis
- (b) Ascariasis
- (c) Trichiniasis
- (d) Uncinariasis
- (e) Strongyloidosis
- (f) Dracontiasis
- (g) Trichocephaliasis.

(a) **Filariasis.**—This disease is due to the presence in the lymphatics, connective tissue and serous cavities, of very slender nematode worms, the larvae being present in the circulating blood. Obstruction of lymphatic channels finally results, with elephantiasis, chylous ascites, and other manifestations.

**THE PARASITE.**—The *Filaria bancrofti* is the one best known and responsible for most of the serious manifestations of filariasis. The *Filaria loa* is of less importance and other varieties cause no very serious symptoms in man, so far as known.

Certain mosquitoes, generally of the genus *Culex* carry the infection to man. The disease is found chiefly in the tropics, but a few cases have originated in the southern United States.

**SYMPTOMS PRODUCED BY THE FILARIA BANCROFTI.**—There may be a long latent period, during which the infection is recognizable only through the embryos in the blood. The reasons for the development of symptoms are not clear, but they may appear if the affection be a severe one, or possibly after injury to the worm, or because, according to Manson, "of the presence of the immature products of conception," blocking of the lymph channels resulting.

Filarial fever is the first symptom of infection, with weakness, and anemia, itching, and, sooner or later, signs of the local effects of the blocking of the lymph channels, elephantiasis, hematochyluria, chylous ascites, filarial abscess, etc.

The enlargement of the scrotum, vulva, etc., but more especially the legs (75 per cent.) is spoken of as elephantiasis. This results

after repeated attacks of the so-called "elephantoid fever." The skin is rough and enormously hypertrophied, with dilated lymphatics and enlarged lymph nodes. The filaria is not found in certain cases, and there is much doubt as to whether it is actually associated with all cases of elephantiasis.



FIG. 32.—SWELLING FROM CHRONIC LYMPHANGITIS, SIMULATING ELEPHANTIASIS. (Case of Dr. Leonard Freeman.)

*Hematochyluria.*—By the rupture of a lymphatic varix in the bladder wall, as following childbirth, violent exertion, etc., the urine becomes chylous and bloody, the proportions of fat globules and of red cells varying at different times and changing the color from a milky one to that of pale blood. A reddish clot may form and cause much difficulty in micturition. The chyluria is intermittent in most cases. Anemia and great debility result from the long continued drain upon the system.

*Chylous Ascites.*—Chylous ascites occasionally results from filarial disease, but much more commonly in this country at least from some mechanical blocking of the chyle passages.

*Filarial Abscesses.*—Filarial abscesses occur in various organs, and the dead worms may be found in them. Manson states that the disappearance of the embryos from the peripheral blood should lead to the suspicion of abscess, in cases of obscure internal symptoms.

**DIAGNOSIS.**—The occurrence of elephantiasis, chyluria and chylous ascites, and especially in those who have lived in the infected

districts, should lead to investigation of the blood for the living embryo, and of the urine, those found here being non-motile. Chyluria from other cause than filarial disease is rare, but the occurrence of elephantiasis and chylous ascites in this country creates no especial presumption in favor of such infection, and laboratory proof is essential to the diagnosis. Stiles states that the lymphocytes increase to 24 per cent. to 40 per cent. and the eosinophiles to 8 per cent to 18 per cent.

**DIFFERENTIAL DIAGNOSIS.**—This concerns chiefly obstruction to the lymph channels, as by adhesions after surgical operations, by pressure of a tuberculous gland, etc. Although the history may direct us to some extent the laboratory must furnish the proof. The appearance of embryos in the blood during the sleeping period, whether this be night or day, is a very important point in the examination.

**SYMPTOMS PRODUCED BY THE FILARIA LOA.**—This variety inhabits the subcutaneous tissues, giving rise to itching and prickling sensations which come and go, and to edematous swellings which appear transitorily in various parts of the body. The "Calabar swelling" of Africa is thought to be the result of infection by this parasite. Manson believes that the *Filaria diurna* is the larva of the *Filaria Loa*.

(b) **Ascariasis** (*Ascaris Lumbricoides*).—This worm roughly resembles the ordinary earth worm in appearance, but may be 25 cm. or more in length, the female being the larger. The body is reddish gray or yellowish in color, tapers from the center to the ends, with four longitudinal bands, and transverse marking. It is the most widely distributed human parasite.

The parasite inhabits the small intestine, but may migrate to the colon, to the gall-bladder, occasionally causing jaundice by blocking the common duct, and to the stomach. It may thence crawl out of the mouth, as I have seen after anesthesia and in typhoid fever, or engage in the larynx or other orifice, such as the perforations occurring in diseases of the digestive tract. A "worm abscess" may result. But a few worms are present in most cases, but they are found rarely in masses in the bowel.



The ova are passed in the stools and gain entrance to the human stomach by means of drinking water or food.

**SYMPTOMS.**—There are no characteristic symptoms of infection with the round worm, but when they are present in large numbers, gastro-intestinal catarrh, colic and even intestinal obstruction may occur. Various nervous disturbances may be noted in children, and a secondary anemia with eosinophilia may occur.

**DIAGNOSIS.**—This depends upon finding the worm or upon demonstration of the eggs in the feces. The fact that the parasite may invade the nose, ear (perforation of the drum), lachrymal duct, trachea, larynx, bronchi, gallduct, pancreatic duct, peritoneum (intestinal perforation), bladder, or appear at the external surface of the body through various fistulous tracts should not be overlooked.

**Oxyuriasis** (*Oxyuris Vermicularis*: Pin Worm, Thread Worm)—This is a very common parasite, inhabiting the large bowel, but especially the rectum, frequently wandering to the vagina in young girls. The habit of the female worm of wandering out from the anus at night accounts for the itching, burning and redness noted. Auto-infection occurs if the child crushes the female in scratching, and the eggs lodge under the nails, since they are thus easily conveyed to the mouth. The direct infection takes place by means of food, water, flies, etc.

**SYMPTOMS.**—These are the vague symptoms of indigestion, when they exist beyond those caused by local irritation. Grinding of the teeth during sleep is attributed by the laity to the infection.

**DIAGNOSIS.**—This is made by finding the worms, either in the feces or wandering outside the anus or in the vagina, especially as the child falls asleep at night. The eggs are easily detected in the feces. Stiles states that the negative examination for the eggs is of little value.

(c) **Trichiniasis.**—This is due to intestinal infection with the *Trichinella spiralis*, the embryos being found encapsulated in the muscles.

The rat is the normal host of the parasite, and the frequency of rats about slaughter houses has much to do with the propagation of trichiniasis, since hogs eat rats, rats eat offal of hogs, and each animal

is cannibalistic. The infection in man occurs from eating infected pork, not sterilized by proper cooking, and is thus accidental. One or two per cent. of American hogs have been found to be infected.

The adult worms live in the small intestine, appearing like fine glistening threads, 2 or 3 mm. in length. The embryos wander generally in the lymph or blood stream, reach the striated muscle fibers, and become encysted, coiled up spirally in an oval capsule. They may remain alive for many years, and if swallowed by man or other host, develop in two days into the mature worm.

**SYMPTOMS.**—After having eaten of infected pork, often when a company of people have eaten underdone pork, sausage, etc., as at some festival, symptoms develop in from two to five days,—nausea, vomiting, diarrhea, and cramp-like pain. If the embryos are few no recognizable symptoms may develop, although in endemics the milder cases may be identified through recognition of the severer ones. The diarrhea may be intense and accompanied by much pain and weakness. After eight to fifteen days, with the migration of the embryos, edema of the eyelids and face occurs, with general myositis, due to the invasion of the striped muscles. The predilection of the embryos for the diaphragm and intercostal muscles makes respiration very painful and may thus be the cause of death. The considerable fever, rapid pulse, mental dulness, insomnia, delirium, sweating, dry tongue, tremor, occasional albuminuria, and history of diarrhea often lead to the diagnosis of typhoid. The soreness of the muscles—intense in cases of grave infection—is a characteristic feature. The swelling in the visible muscles is chiefly noted near the tendinous insertions. The tendon reflexes are lessened or even abolished.

**DIAGNOSIS.**—The history of illness with the symptoms described, especially in several persons, after eating underdone pork, is of importance. The encysted larvæ may be detected in remaining portions of the pork, and the silvery thread-like worms may be isolated from the stools, especially if the diarrhea continues. The dejecta are diluted with water and the worms recovered from the bottom of the glass after shaking and pouring off the supernatant portions. In bits of muscle removed from the arm of the patient, the embryos may be detected in the third week. A high leukocytosis with an ex-

treme eosinophilia is very suggestive. The latter may persist for some time after recovery. Herrick, Janeway, and others have found the embryos in the blood after the sixth or seventh day. The method is not always successful.

**DIFFERENTIAL DIAGNOSIS.**—If the procedures outlined above be carried out there should be no great difficulty in excluding typhoid fever, and the Widal reaction may still further reduce it. Cholera is occasionally suggested by the profuse diarrhea, but the examination of the stools for the comma bacilli should be decisive.

**PROGNOSIS.**—In nearly 15,000 collected German cases the mortality was 5.6 per cent. (Stiles). Packard found the mortality to be over 44 per cent. in 357 reported American cases. The outlook is better if the infection be light and if diarrhea have carried off many of the worms. It is better in children than in adults. Severe dyspnea, high fever, coma, delirium, and great weakness are of grave omen. The patients in the favorable cases may not fully recover for several months.

(e) **Uncinariasis** (Hookworm-Disease).—The *Anklyostoma duodenale* is the hookworm of the Old World. Stiles found in America a new variety, the *Necator americana*. The worms have the same general characteristics, and it has been found that each to some extent invades the field supposed to belong to the other. Oliver states that the American variety has been found in the interior of Africa, and it may have been introduced here by the African slaves.

The hookworm is a small nematode parasite, 8 to 18 mm. in length, living in the ileum, but occasionally found in the duodenum or even in the stomach. The ova pass out with the feces and develop into rhabditiform embryos, which finally become encysted larvæ, and reinfect man, either through the skin or by being swallowed with food, water, etc. The passage of the larvæ through the skin in the so-called "ground itch" is presumably much the more frequent method of infection, although entrance through the skin to the circulatory system, passage to the heart, the lungs, to the air passages up to the larynx, thence to the stomach through the esophagus, and thence to the intestines seemed at first so round-about a method of introduction as to seem improbable. The development is thus with-

out an intermediate host. The parasites attach themselves to the mucous membrane of the small intestine, and cause damage by the hemorrhage produced by the wound. The bleeding is greater because of the secretion by the worms of a substance which inhibits coagulation. In the examination of the mucous membrane one may see the bites, often surrounded by hemorrhage. A chronic intestinal catarrh is present, and even a chronic interstitial inflammation with thickening and constriction has been noted. The changes in the heart, spleen, and other organs are chiefly due to the secondary anemia of the infection.

Hookworm-disease is found in those living in warm, damp climates, favorable for the development of the embryos, and under conditions in which the fecal discharges are permitted to contaminate the surface of the soil, and the skin of the subjects gives the opportunity for the embryo to penetrate it; in other words, it is essentially a disease of bare-footed people, without hygienic conveniences. Those coming in contact with the damp earth,—farmers, miners, etc.,—are naturally more liable to infection. This disease is common in the operatives of European mines and tunnels, but practically unknown in the miners of the northern half of the United States, and rare in those of the South. The South Atlantic and Gulf States, Cuba, Porto Rico, and the Philippine Islands have furnished most of the cases studied by American clinicians. Because of the characteristic anemia the disease is called by such names as Southern anemia, Egyptian chlorosis, etc.

**SYMPTOMS.**—Anemia, weakness, perverted appetite, dirt-eating, etc., palpitation, dyspnea, vertigo; and in children, in whom it is most frequent, delayed development, are the common manifestations of the severer infections. In many cases the papular dermatitis, noted especially between the toes and on the sides of the feet, may be found, and represents the initial point of infection. In the well-marked cases the waxy skin, pallid mucous membrane, the blank stare, noted by Stiles, slow mental processes, dilated heart, edema of the legs, weakness and lassitude, “pot belly,” and the evidences of lack of development of the genital organs are most striking. Yet many persons in apparent health, in the infected areas, pass the hook-

worm eggs in the stools. In the well-fed, with moderate infection, no symptoms appear, while in those living upon a less substantial diet, with some degree of infection, but more particularly with severe infection, the disease presents the symptoms noted above.

**PHYSICAL EXAMINATION.**—Ashford, King, and Gutierrez speak of pain and tenderness in the epigastrium, as being quite constant and very suggestive. Enlargement of the liver and spleen is found in severe cases. Hemic murmurs are common. The muscles are soft and flabby.

In the feces the ova are found with great ease and in great abundance. Blood and mucus may be present macroscopically. In the Porto Rican work the hemoglobin averaged less than 50 per cent., and I have seen it very low, even in cases imported into Colorado from the South. The red cells present the changes usual in secondary anemia. Leukocytosis is not a feature of the disease, but a moderate increase in the eosinophiles is commonly present and may be taken as indicating a good resistance to the toxins of the parasite.

**DIAGNOSIS.**—Marked anemia in those living in or recently from infected areas should suggest the examination of the stools for the ova, upon which the positive diagnosis must be based in most instances. The adult worm may be found also, especially after giving an anthelmintic. Motile larvæ develop in warm, moist earth in two or three days, and may be easily seen with low magnification. The staining of blotting paper by the blood in the feces is suggestive when it occurs.

**DIFFERENTIAL DIAGNOSIS.**—This must consider pernicious anemia, the leukemias, and other blood diseases. Aside from the distinguishing features of these affections one may confidently rely upon the laboratory examination of the feces for the eggs of the parasites.

**PROGNOSIS.**—This is excellent in American patients under proper care, the infection being very amenable to treatment by thymol and other anthelmintics. In ignorant subjects, not under supervision, the outlook is less favorable. The Porto Rican Commission believed that 30 per cent. of the deaths in that island were due to uncinariasis, but of course this mortality occurred in untreated cases.

(e) **Strongyloidosis.**—The *Strongyloides intestinalis* is found in

the feces of many persons living in the tropics, and is especially common in parts of China. It was formerly thought to be the cause of Cochin-China diarrhea, but its pathogenicity is not established. Strong believes that a heavy infection with this parasite may be responsible for a catarrh of the small intestine with diarrhea.

**DIAGNOSIS.**—This depends upon the finding of the rhabditiform embryos in the diarrheal stools. I have reported one case in a Chinaman, supposed clinically to be amebic dysentery, in which multitudes of the embryos were found in the microscopic examination of the stools. Few cases have been reported in the United States.

(f) **Dracontiasis.**—This is caused by the *Dracunculus medinensis* or guinea-worm, a nematode inhabiting the connective tissue. The female adult is over two feet in length, and very slender. The male is not absolutely identified. Infection is thought to occur by drinking water containing the intermediate host, a small crustacean, the cyclops.

The infection is found chiefly in Africa, but has been introduced by African slaves into South America. Imported cases have been reported in the United States.

The impregnated female burrows in the connective tissue, and after many months reaches some part of the leg, where it is felt under the skin as a slender cord. Finally a small ulcer forms upon a swelling near the ankle, and the worm may present its head at the orifice or leave its host spontaneously. An abscess may form if the worm dies. Sharp febrile reaction may be present.

**DIAGNOSIS.**—This depends upon the finding of the worm or of the embryos discharged from the orifice at the base of the ulcer. Eosinophilia may be noted.

(g) **Trichocephaliasis.**—This disease is the result of infection by the *Trichuris trichuria* or whip-worm. This is a common and widely distributed intestinal parasite, especially in warm countries, but productive of no especial symptoms in most cases. The worm inhabits the cecum and colon preferably. It is two inches or less in length, somewhat resembling a whip in shape.

**SYMPTOMS.**—Possibly anemia, diarrhea, and debility may result from severe infection with this parasite.

**DIAGNOSIS.**—This is made by the finding of the parasite or its ova in the stools.

Other nematode infections are so rare or of so little import, that the reader is referred to the special works for their further consideration. I have known of two instances in which a short, thick worm has been found in the subcutaneous tissues in the region of the neck, in this state, but have not been able to learn its identity.

### III: TENIASIS: INFECTION BY CESTODES

The tapeworms cause disease in man in two wholly different ways: First, by infecting his intestinal tract as a tapeworm; and, secondly, by infecting other portions of the body in the larval state.

The cestodes are long, flat worms, with small head and a series of from 3 or 4 to 1,000 segments (proglottides), varying greatly in size, number, shape and appearance, with the different varieties of worm. The head (scolex) is furnished with suckers or hooklets for attachment to the intestinal wall, varying with the species. In the common tapeworm male and female sexual organs are present in each segment, but only the more mature segments, remote from the head and neck, have ripe ova.

Man is the normal host for the tapeworm stage of the beef and pork tapeworms. The other two of chief importance in human medicine, the fish tapeworm and the dwarf tapeworm, are found in the human intestine, but also in that of lower animals. Stiles states that ten other species of cestodes are occasionally found in man, but are of comparatively slight importance.

The beef tapeworm has been regarded as vastly more common in America than any other, the pork tapeworm being rare, while the fish tapeworm is practically seen only as imported from the region around the North Sea. The recent reports of the finding of the dwarf tapeworm in various parts of the United States lend color to the statement of Stiles that this affection is fairly common but not generally recognized. With the exception of one imported case of infection by the *dibothriocephalus latus* every tapeworm that I have known of in Colorado has been of the variety *T. saginata*. The

*Tenia solium* is most dangerous, because of the occasional development of larvae in the brain, eye or other tissues. The *dibothriocephalus latus* causes an anemia so severe as to be confused with pernicious anemia, but the remaining varieties are much less harmful.

**Species of Cestode.**—*T. SAGINATA* (Fat Tapeworm; Beef Tapeworm).—This cestode is very widely distributed. It is one of the largest of the tapeworms, reaching commonly a length of four or five meters, and at times even more than ten meters. It inhabits the small intestine of man, being attached by means of the suckers upon the head, there being no hooklets. The ripe segments are 16 to 20 mm. long, and 4 to 7 mm. in width, and are commonly passed from the bowel singly or in small groups. They may be seen to move in the stool, so that they may be easily distinguished from the strings of mucus and other deceptive objects which patients so often mention.

The ripe segments, or more frequently, probably, the ova from them, are swallowed by cattle, and the beef from such animals is found to be infected with the *cysticercus bovis*, the larval form of the parasite. The eating of raw or too lightly cooked beef so infected gives rise to the tapeworm infection in man. The tongue especially contains the *cysticerci*.

*T. SOLIUM* (Pork Tapeworm).—This, the armed tapeworm, so-called from its double row of hooks for attachment to the intestinal wall, is much smaller than the *T. saginata*, perhaps averaging 3 m. in length. The largest segments measure about 12 mm. in length by 6 mm. in breadth. The ova pass out with the feces, and develop only upon being taken into the stomach of a suitable host—the hog, normally, but occasionally man. The digestive process sets the embryos free, and they pass through the intestinal wall, become encysted in the muscular or other tissues after developing the tapeworm head, and remain quiescent until an opportunity arises for further development, after entering the stomach of a suitable host (man). The pork tapeworm in man is said to give the larval form of infection to its host in some instances through the passage of mature segments upward into the stomach, as in severe vomiting.

The infection is brought about in man by the ingestion of infected pork not sterilized by thorough cooking. Mild curing or pick-



ling methods do not suffice to kill the embryos in the central portion of the meat. The frequency of infection by this form of cestode amongst those peoples accustomed to the use of raw pork or pickled but uncooked pork, and the rarity amongst others, has long been recognized.

Persons infected with *T. solium* should be promptly and vigorously treated, because of the danger of its producing the cysticercus infection mentioned, and because of the fear that those associating, and especially sleeping with them may become infected through accidental ingestion of the discharged ova.

In the case of *T. solium* the segments passed from the bowel are likely to be joined together in a fragment several inches in length.

*HYMENOLEPIS NANA* (Dwarf Tapeworm).—Stiles believes this to be the most common tapeworm in America, but it is rarely discovered unless especially sought for because of its minute size. The eggs swallowed accidentally develop in the villi of the intestine into larvae, which in turn develop into the adult worm in the intestine, no intermediate host being necessary.

The presumption being at present that the human parasite is identical with that of the rat and mouse, the infection probably comes from the food exposed to the rodents and contaminated with their feces. Thus Amessee reports the case of a child living in a poor New York tenement, in whom a heavy infection with the dwarf tapeworm existed. Amongst the crackers on which it was feeding were the evidences of contamination by mice.

It is more common in children than in adults. In several hundred children examined by Stiles in the Southern Atlantic States, from 2 per cent. to 4 per cent. were infected.

The worm varies from 5 to 45 mm. in length, with 100 to 200 segments. It is found especially in the upper two-thirds of the ileum.

*DIBOTHRIOCEPHALUS LATUS* (*Tenia Lata*).—The broad or fish tapeworm is found particularly in the regions about large bodies of water where the larval form is found in the pike, burbot, perch, salmon, etc. Japan and parts of Europe bordering on the Baltic are the especial regions of infection by this worm, but it is found in most

countries where fresh fish are largely used, and especially where, as in Japan, they are eaten raw. Many instances of infection by this parasite have been reported in the United States, chiefly amongst immigrants from some of the infected regions mentioned. Warthin reports the infection amongst the Swedes and Finns of the mining population in northern Michigan; and the fish of Lake Superior are suspected.

The adult parasite is an enormous worm reaching at times a length of ten meters and a breadth of 20 mm. The great transverse breadth of the anterior segments and the darker color of the central spot (uterus) render its distinction from our ordinary tapeworms easy. The parasite is known to develop with great rapidity in the intestine.

For a description of other tapeworms, less frequently found in man and of less clinical significance, the reader is referred to the larger works.

**Symptoms of Intestinal Teniasis.**—Only in exceptional cases are there symptoms which would attract attention to the infection, and the finding of segments in the feces is generally the first intimation of the presence of the parasite. Ravenous appetite, loss of appetite, digestive irregularities, nausea and diarrhea have been occasionally noted, but are never sufficiently distinctive to justify more than a suspicion of the cause, which must be established by examination of the feces for ova, or by the finding of segments of the worm. The administration of calomel, with a saline afterwards, will commonly result in the detachment of segments, upon which the diagnosis may be based.

Mechanical obstruction is a very rare feature of teniasis, and no intestinal lesions are produced ordinarily. The loss of nutriment because of absorption of a portion by the worm must be trivial. Peculiar sensations of movement and of discomfort in the bowels are frequently mentioned. The action of the toxins eliminated by the worm is probably by far the most serious feature of teniasis, if we except the possibility of the development of cysticerci in the case of *T. solium*. An anemia closely resembling the pernicious form is not infrequently found in the Baltic provinces, as a result of infection

with the fish tapeworm. Prompt recovery sometimes occurs upon the expulsion of the parasite. Convulsions, palpitation and other symptoms of nervous origin are not very rare.

**Diagnosis.**—This is easily established by examination of the feces for segments of the worm and for the ova. The frequency with which patients bring shreds of mucus, etc., to the physician justifies him in demanding to see the parasite or ova before the institution of treatment. The rarer infections demand the assistance of a helminthologist.

**Prognosis.**—This is good excepting in the severer infections with the fish tapeworm, in which death may occur from anemia, even after the worm is expelled.

#### SOMATIC TENIASIS

This results from infection with the larval stage of tapeworms. By far the most important form is that resulting from the development in man of the larvæ of the *tenia echinococcus*. Occasionally infection is found with the *cysticercus* of the pork tapeworm, but other forms of visceral infection are uncommon.

#### ECHINOCOCCUS DISEASE

*T. echinococcus* (*E. granulosus*, Stiles) is a minute tapeworm of the dog, dingo and similar animals, living in the small intestine. The ova reach the intermediate host (sheep, cattle, hogs, etc.) and occasionally man (accidental host) through contamination of water and food with the egg-bearing feces. In countries where infected dogs and sheep come into contact with man, the disease is common, notably in Iceland and in Australia. The frequency of the infection in parts of Germany and among the Icelandic immigrants in Manitoba is well recognized. The only two cases coming under my observation occurred in Italian miners, the source of infection being unknown.

The eggs from the mature terminal segments having reached the human stomach, the six-hooked embryo escapes from the shell, bores

into the liver or other organ, and begins the development of the vesicle, filled with water, which gives it the name of "bladder worm." The inner membrane is spoken of as the germinal layer or endocyst, the outer as the capsule. A firm connective tissue furnished by the host may lead to the arrest of development of the cyst (acephalocyst, sterile cyst). Normally the cyst forms daughter-cysts and even granddaughter-cysts from the germinal layer (endogenous echinococcus). Smaller exogenous cysts are occasionally seen in man. The multilocular echinococcus is classed by Stiles as a subspecies. The mother cyst may suppurate or be destroyed in other ways. The collection of cysts and daughter-cysts with the surrounding fibrous capsule gives rise to an irregular nodular tumor, sometimes of great size.

The fluid has a startling resemblance to drinking water, with a density of 1.005 to 1.015, and contains traces of sugar and sodium chlorid. With the fluid may appear, upon draining the cyst, scores or hundreds of daughter-cysts, ranging upward to the size of a marble, and containing the same clear fluid. In man more than half of all the cysts are located in the liver, the lung and kidney being the next most frequently infected organs; but the cysts may develop anywhere in the body. If the parasite dies the cyst collapses to some extent, and the contents may become opaque and gelatinous. The heads or hooklets should be sought for in the fluid. Death of the cyst may result in the calcareous degeneration of the remaining wall. Rupture into the lung or a blood vessel may be very serious. Suppuration with external discharge is more favorable, and occurs especially in the hydatid cyst of the liver, being at times confused with true abscess of the liver.

**Symptoms.**—The hydatid cyst ordinarily manifests its presence only by its gradual growth, causing symptoms by its pressure in accordance with the location. There may be no complaint excepting of the mechanical inconvenience, as in the case of one patient of mine, with an enormous liver, who protested daily that he was not sick, but could not work on account of his protuberant abdomen. The liver and the omentum were filled with cysts.

In the brain the symptoms produced are those of tumor. I have

seen profound "black" jaundice from compression of the common bile duct, and marked dyspnea from the compression of the lung. Hemoptysis may occur. A feeling of pressure and tension in the abdomen and pelvis may be present, pain being noted in case of severe pressure. Ascites may result from pressure upon the portal vein.

If the cyst become infected with pus-producing organisms, symptoms of sepsis develop, and the signs of an abscess. Rupture of a cyst into the lung may practically drown the patient; if into the biliary tract, suppurative cholangitis with jaundice may develop. The vena cava inferior has been perforated, with death from embolic plugging of the cardiac orifices or the pulmonary arteries by the daughter-cysts. The contents of a cyst may be evacuated by rupture into a bronchus with expectoration of fluid; by rupture into the bowel, with passage of the contents into the stool; or into the urinary tract, or externally. Rupture of a cyst internally is occasionally followed by an intense urticaria, attributed to a toxin found in the fluid.

Physical examination ordinarily shows only a tumor, generally smooth or nodular, insensitive, and occasionally giving the characteristic fremitus upon tapping with the fingers as for the detection of ascites. The physical signs in the lungs are those of a growth—flatness, displacement of other organs, and interference in various ways with the usual sounds of respiration.

**Diagnosis.**—The history of exposure in one of the zones of infection should be considered. A large tumor without symptoms other than those due to its mechanical interference with surrounding organs, especially if it be insensitive, and unaccompanied by adenitis, should suggest exploration with a needle. The finding of a clear watery fluid raises a presumption in favor of hydatid cyst; the presence of sugar and sodium chlorid in the fluid is almost certain to indicate it, while certainty requires the finding of cysts, scolices or hooklets. The diagnosis of multilocular echinococcus infection depends upon the finding of the so-called "colloid cancer" in the liver, this being the term applied to the multilocular cyst in this region, before its true origin was recognized. This form is practically un-

known in America, with the exception of a very few imported cases.

In regions where hydatid disease is not endemic, the diagnosis is presumptive only, whether the infection invade the brain, lung, pleura, liver, omentum, kidney or other organ, unless the hooklets, scolices or cysts be demonstrated. A high degree of probability may exist in a given case without such proof, and in Iceland and parts of Australia the disease is so common that a presumption in its favor exists in all obscure internal diseases. Ghedini has introduced a complement fixation test, but its utility is not entirely established.

**Prognosis.**—This depends entirely upon the possibility of surgical intervention, and this in turn depends largely upon the location and severity of the infection. I have seen the disease recur five times after what appeared, each time, to be successful intervention, new cysts developing rapidly as soon as the removal of pressure by operation permitted. The danger of aspirating cysts in the lung without preparation for immediate operation, in case of flooding of the bronchi, should be mentioned.

#### CYSTICERCUS CELLULOSAE

Infection with the bladder worm of the *T. solium* is not often met with in America. The cyst may develop anywhere in the body, occasionally sub-cutaneously. Many instances of development in the brain and in the eye have been reported, perhaps because the infection is more readily recognized in these localities. The severe infections are thought by Stiles to result from the regurgitation of perhaps an entire segment from the intestine into the stomach, with the simultaneous letting free of a multitude of embryos through the action of the acid gastric juice.

**Diagnosis.**—This is extremely difficult on account both of the rarity of the disease and the lack of definite signs and symptoms. A reasonable degree of probability is as much as is attainable in the diagnosis in most instances.

**Prognosis.**—Cure may result from operation upon a brain tumor found at operation to be a cysticercus, and the cysts have been re-

moved from the eye. In many cases the cysts in other localities lie dormant for many years.

The rare infections by the larval forms of the beef tapeworm and certain other cestodes are of helminthological rather than clinical interest.

#### IV. MISCELLANEOUS INFECTIONS

For a consideration of the caterpillar rashes and of infection with various mites, ticks, leeches, the harvest-bug, fleas, lice and bedbugs, the reader is referred to the special works.

The danger may be not only that from the irritation produced, as by vermin, but from the carrying of infection, as in the case of *Dermacentor occidentalis* in the tick fever of the Rocky Mountains, and various other infections already considered.

**Screw-worm.**—Numerous instances have been reported from the Southwest of the finding of the larvæ of the *Comptosia macellaria* in the nostrils and in wounds. In those sleeping out-of-doors this easily happens from the opportunity offered for the fly to deposit its eggs. The screw-worm develops in a few hours, burrows into the tissues and may cause serious symptoms for a week before pupation. Individuals with an offensive nasal catarrh and particularly with atrophic rhinitis especially attract this fly. The infection is decidedly serious, about 70 per cent. of the cases resulting fatally (Maillard).

**House-fly.**—The larvæ of the house-fly and other dipteræ are occasionally passed by the bowel, having been swallowed by accident. In some cases flies have oviposited upon the prolapsed rectal mucous membrane and the larvæ have developed within the rectum and been later expelled. In a case of V. R. Pennock's, the dead larvæ of the flour-beetle were passed in the stools on several occasions. The occurrence of maggots of various kinds in the discharging ear and in foul wounds is happily less frequent than before the days of antisepsis.

In general we may say that the not infrequent reports by patients and especially by neurotic women of the passage of strange worms, insects, larvæ, etc., are to be scrutinized with great suspicion.

## E. INFECTIOUS DISEASES OF DOUBTFUL OR UNKNOWN ORIGIN

### I. SMALL-POX

**Definition.**—Small-pox or variola is a highly contagious, infectious, epidemic disease, characterized by a typical fever and a pustular eruption.

Practically all unprotected individuals exposed to the contagion of small-pox take the disease, and neither age, race, sex nor other conditions offer protection. The colored race and, in general, those lower races of mankind who have escaped contact with civilization are especially susceptible when exposed. Pregnant women often suffer from it. The disease is endemic in large centers of population, breaking out under favorable circumstances to become epidemic. The child in utero may have the disease and be born with the scars, and I have seen eruption present at birth.

The protozoön described by Councilman is found in the deeper layers of the epithelium, and has been found so constantly by so many observers that it is widely accepted as the cause of the disease. The infectious agent is thrown off from the body in crusts and scales, and probably in the expired air, exhalations from the skin and the excretions. The disease is communicable before the eruption appears and until the skin has become healed and the body and clothing disinfected. The crusts and scales are presumably the most active agents in transmission, and through them the disease may be carried by a third person uninfected, and by fomites. An epidemic in Denver started 13 days after the receipt of a letter from a patient with small-pox in Manila, distant a third of the circumference of the globe. Baled rags, infected clothing tightly packed away, carpets, etc., may carry the infection even for years. The blood, lymph, pus from the vesicles, and discharges from the mouth and nose may carry the disease. In several instances, it has undoubtedly been air-borne for the distance of a city block. Flies and possibly other insects may aid in its dissemination.

**Varieties.**—It is common to divide small-pox into forms as follows:



Variola Vera	$\left\{ \begin{array}{l} (1) \text{ Discrete small-pox} \\ (2) \text{ Confluent small-pox} \\ (3) \text{ Hemorrhagic small-pox} \end{array} \right.$	$\left\{ \begin{array}{l} (a) \text{ Purpura variolosa} \\ (b) \text{ Variola pustulosa} \\ \text{hemorrhagica} \end{array} \right.$
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**Variola Modificata.** 1. Varioloid (Small-pox modified by vaccination). Variola sine eruptione and abortive small-pox will be described separately.

**Symptoms.**—The period of incubation is from five to fifteen days, generally twelve or thirteen, but less in the more malignant forms. The first symptoms of the disease come with an acute onset, chill, headache, severe backache, vomiting and fever. The chill often recurs in adults and is replaced by convulsions in children. The severity of the backache arouses the suspicion of the physician familiar with small-pox, even in the absence of known exposure.

The fever rises to  $104^{\circ}$  to  $106^{\circ}$  and remains high with slight remissions until the appearance of the eruption. The respiratory rate and pulse rate are correspondingly elevated, and the pulse is full and bounding, excepting in the malignant forms, when it is feeble, irregular, and often intermittent.

Flushed face, injected conjunctivæ, coated tongue, delirium, pharyngitis, thirst, precordial oppression, nausea, vomiting, insomnia, even coma, and intolerable headache, and especially backache, are features of the severer cases. Excepting for the palpable spleen often found, physical examination is practically negative. The bowels are constipated, the urine scanty, high-colored, and generally albuminous. If the patient be so overwhelmed that he cannot stand, a malignant form of small-pox is to be feared. Hematuria signifies a hemorrhagic form.

Women commonly flow as if at the period of menstruation, and abortion frequently sets in at this time in pregnant women.

Leukocytosis does not appear until later. In variola siderans the patient dies before the eruption appears, overwhelmed by the onset of the disease, and death often occurs in purpura variolosa at this time.

**INITIAL RASHES.**—During the second and third days after the

chill we may note the accidental rashes of small-pox in a small percentage of the cases. The roseolous form is most common, distributed upon the face, trunk and extremities. The erythema is seen less frequently and may cover even the entire body, being commonly fainter upon the legs. This form is often associated with numerous purpuric spots, particularly abundant over the lower abdomen, genitals, and inner surface of the thighs. It may also be found in the axillary regions, and below, upon the lateral and anterior aspects of



FIG. 33.—DISCRETE SMALL-POX. Tenth day.

the chest, and the inner arms. This type of rash may remain after the exanthem appears and is of grave significance.

At the end of about three days, the fever falls, headache, back-ache, sleeplessness and discomfort abate, and the eruption shortly begins to appear. Upon the character of the exanthem depends chiefly the classification of the different varieties of small-pox.

**DISCRETE FORM.**—With the end of the third or beginning of the fourth day of this disease, the eruption appears, generally first at the edge of the hair upon the forehead, and at the anterior surfaces

of the wrists. I have seen it first upon the abdomen. For two or three days it spreads over the body and limbs, being slightly more advanced upon the face, which has been first involved in most cases. The typical eruption gives the impression that practically all the pocks are in the same stage of evolution, but in the milder epidemics of the past dozen years this statement does not hold. The eruption may be especially abundant at the lines of the waist and collar. With the fall of temperature which occurs at the time of the eruption the patient in mild cases feels almost well, and may wish to get up.

The pock at first is a reddish macule, slightly indurated, one to three millimeters in diameter, and becoming pale if the blood be pressed out of it by the finger. During the next twenty-four hours the pock becomes harder and the "shotty" feel of the eruption along the edge of the forehead is almost pathognomonic. Shortly a minute vesicle with clear contents appears at the center, gradually spreading until it is of the size of the papule, and often umbilicated, because of the rapidity of development at the periphery. A red areola is present and some little swelling of the surrounding skin. The fluid within is in separate pockets, which cannot be evacuated by a single puncture as is the case in varicella.

The fluid gradually becomes more opaque, then yellowish, and at maturation, about the sixth day of the eruption, fills the pustule so that the umbilication is obliterated. By the tenth day of the disease retrocession begins, the pustule becoming less tense, a secondary umbilication perhaps appearing with the resorption of the contents and hard, yellow, brown or black crusts finally develop, separating toward the last of the third week. If the pustules have implicated the lower layers of the skin, permanent scars result (pitting), but this is generally insignificant in discrete small-pox.

Discrete small-pox is fortunately much more common than the severer forms, gives rise to but little scarring, and has but slight mortality.

**CONFLUENT FORM.**—The distinguishing feature of confluent small-pox is the coalescence of the pocks, this occurring early in the development of the eruption, and in the region where the eruption is most abundant, commonly the face. The degree of confluence upon

the face gives the best criterion for the estimation of the severity of the attack and its danger to life. The hands, feet, wrists and ankles often present a confluent eruption. The severity of the attack in a given individual in no wise depends upon the character, whether discrete, confluent or hemorrhagic, of the disease from whom the con-

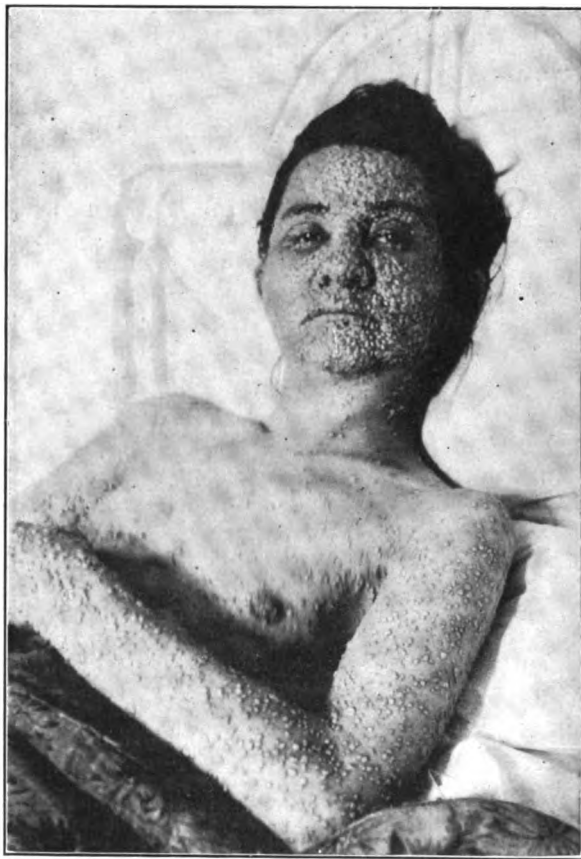


FIG. 34.—CONFLUENT SMALL-POX. Ninth day.

tagion came. I have known fatal hemorrhagic small-pox to arise from exposure to a very mild case of varioloid. A greater or less degree of individual immunity must be the deciding factor in this problem.

Pustules appear upon the hard and soft palate in many cases, often very early and frequently rupturing early and presenting

whitish ulcerations. The eruption is commonly present upon the corneal, nasal, buccal, laryngeal and tracheal mucous membranes also, and not infrequently upon the mucous surfaces of the genital organs and of the anus. In these regions the roof of the pustules frequently macerates and breaks through, leaving distressing ulcerations, varying in their symptomatology with the location. The eruption upon the palms and soles is fairly characteristic of small-pox as contrasted with varicella; and the pock, deep in the horny layers, develops without much elevation of the surface in many cases. The pustule may leave, in this region, upon absorption, a rounded cicatrix, which must be picked out with the knife. The pustules occasionally seen under the nail are very painful, and patients not very ill may scrape away the nail and relieve the tension.

Great itching may be complained of during desiccation. The hair commonly falls and the nails may separate. Extensive areas where confluence has been complete may heal by granulation like other surface ulcerations. The thick skin of the palms and soles may come off as a whole. As the cicatricial tissue in the severely involved areas contracts in the healing process, irregular tension upon parts of the skin causes the lower eyelid to evert, and even disturbs the normal position of the lips. The facial expression may be thus greatly modified.

During the secondary fever the temperature commonly rises to about the height of the initial fever, and a marked polynucleosis is present. Delirium may be noted. Secondary infection may occur in the pustules, streptococci being commonly present, and septicemia may develop.

In normal cases the secondary fever subsides in three or four days so that the temperature is near the normal line by the fifteenth day in cases of moderate severity.

In the confluent form the symptoms of invasion are more severe, the eruption often appears a half day earlier than in the discrete form, and its spread is more rapid. More distress is noted as the result of the presence of the eruption in the nares, mouth, larynx, pharynx, etc. Less of a remission is seen at the stage of eruption. The coalescence of the pustules gives rise to what are practically ex-

tensive abscesses under the skin of the face. The yellowish appearance of the countenance is a striking characteristic. Great swelling and disfigurement, severe pharyngitis, extensive cervical adenitis, offensive discharge from the nose, corneal ulceration, salivation, hoarseness, abscess of the tonsils, necrosis of the cartilages of the larynx, diarrhea, delirium, subsultus, involuntary discharges, septic involvement in distant organs, hyperpyrexia, exhaustion and death may be features in the severer cases. Probably the most repulsive sight which disease offers is the swollen, discolored, necrotic, distorted, suppurating, perhaps hemorrhagic countenance of the patient with confluent small-pox. If we add to this the delirium, general helplessness, loathsome surroundings and the foul stench, the catalog of horrors is complete. If recovery occurs, an irregular fever with or without the various complications may prolong the period of convalescence for weeks. Confluent small-pox is seen almost solely in those unprotected by vaccination or by a previous attack of small-pox.

HEMORRHAGIC SMALL-POX.—(a) *Purpura Variolosa* (Purpuric Small-pox).—This is the most malignant and atypical form of small-pox. It affects young adults oftener than children. The incubation period may be only five to eight days, and prodromal aching and malaise may be present. The invasion is often overwhelming in its severity, although the temperature may be moderate. Prostration, small and feeble pulse, precordial distress, vomiting, purging, and atrocious backache are commonly present. A dark erythematous rash, with scattered petechiæ comes out early, often on the second day. The ecchymoses appear upon the face and in the conjunctivæ, perhaps coalescing so that the whole face is swollen, livid and purplish, the edema accompanying the purpura adding to the disfigurement. Bleeding from the gums, nose, kidneys, bowels, stomach and lungs, especially the three former, may follow. Uterine hemorrhage occurs in women, with abortion, if pregnant. Albumin, blood cells and blood casts appear in the urine. Since death always occurs, generally on the fourth or fifth day, the eruption may not appear, or only very incompletely. Councilman states that even in the former cases the histological examination of the skin shows that a universal

confluent small-pox was in the process of development, and that an intense streptococcic septicemia was present in all cases examined by him.

(b) *Variola Pustulosa Hemorrhagica*.—Hemorrhagic pustular small-pox is much more common than the purpuric variety, generally occurring in unvaccinated alcoholics and other debilitated subjects. Although less severe than the last described variety, the hemorrhagic tendency is to be taken as an indication of the severity of the disease.

This variety develops like an ordinary attack of small-pox, up to the time of the eruption, and not until this becomes vesicular or pustular does hemorrhage into the pocks commonly occur. Bleeding may take place from the mucous membrane and ecchymoses may be found between the pocks. The latter often fail to develop into normal variolous pustules. The petechiæ are more numerous upon the legs than elsewhere, in the majority of cases. Bullæ containing blood may appear. The outlook in a given case is closely bound up with the time of appearance of the hemorrhage into the pocks, being graver as the bleeding is earlier. If the hemorrhage does not occur until the pustular stage, recovery may ensue, but most cases die before the ninth day. Hemorrhage may occur in an occasional pock, especially upon the leg, in ordinary discrete small-pox without implying anything further than an accidental complication.

MODIFIED SMALL-POX.—*Varioloid*.—In those who have acquired a partial immunity against small-pox through vaccination, a light form of the disease may occur. The initial symptoms are mild, but few papules occur, chiefly upon the face and hands, the lesions are very superficial, the initial fever subsides by crisis, no fever of supuration occurs, and the course is therefore shorter, and less severe. The incubation period and the invasion may not differ from those of discrete small-pox. There may be so few papules that there is danger of overlooking the disease unless the patient be stripped and examined minutely. Even these few may abort so early as to add further to the difficulties of diagnosis. Since the most malignant small-pox may arise from such cases they demand the most careful investigation.

**VARIOLA SINE ERUPTIONE.**—Variola sine eruptione is rare, occurs in young subjects partially protected by vaccination, and runs a favorable course. The diagnosis is possible only when exposure to small-pox is known, and is then based upon the symptoms of the stage of invasion and the defervescence at the time the eruption should have been expected. Infection may occur if susceptible subjects are exposed, and might clinch the diagnosis.

**ABORTIVE SMALL-POX.**—This term is applied to those mild cases in which the eruption suddenly retrocedes, generally after having reached the vesicular stage. Hemorrhage into the pocks may be noted at this time, without any other signs to denote gravity of the attack. A few pocks especially upon the legs may go on to full development, and absolutely establish the identity of the disease. The variety spoken of by the older writers as "wart pox" or "horn pox" is an abortive type in which the pocks, especially of the face, dry up at the stage of vesication, and become elevated crusts.

**THE BLOOD SYMPTOMS.**—Although in milder forms of small-pox no increase in the white blood cells may be found, in the severer types a polynucleosis is present, 12,000 or 15,000 whites being found at the stage of pustulation. The mononuclear cells are often moderately increased in number. The hemorrhagic forms of small-pox show a greater leukocytosis.

**Complications and Sequelæ.**—The most important complications are those relating to the respiratory tract, and the one causing the greatest anxiety is bronchopneumonia. It is a frequent cause of the fatal result in children. The streptococcus and pneumococcus are the ordinary infective agents.

The bronchopneumonia is often dependent upon a descending infection from the lesions, whether pustular, ulcerative or necrotic, of the larynx and trachea. Lobar pneumonia, pleurisy and empyema are much more infrequent and less directly dependent upon the original disease.

Next in importance are probably the infections from associated pyogenic organisms, which occur in the conjunctivæ, and which in conjunction with the lack of drainage, because of the great swelling of the lids, so commonly caused blindness, notably in children and es-



pecially in the prevaccination days. Otitis media is occasionally seen.

Various septic complications attend the severer types of small-pox, such as endocarditis, pericarditis, suppurative arthritis, parotitis, osteomyelitis and septicemia. Boils, local gangrene (noma), bed-sores, venous thrombosis, dysentery, nephritis, orchitis and diphtheria may occur. Cerebral lesions are occasionally present, giving rise to hemiplegia, aphasia, etc. Paraplegia may result from a focal lesion in the cord, presumably vascular in origin in most cases.

As a sequel of small-pox, we may have neuritis involving the branches of the vagus, possibly raising a question as to a complicating diphtheritic infection. Multiple neuritis is an occasional sequel, and postfebrile insanity of the confusional type may occur.

Contraction of the cicatricial tissue about the larynx after ulcerative processes may affect the voice. A similar process involving the eyelids and face has been mentioned. Deformities in the nares may arise from superficial scar-formation or from deeper necrotic processes.

**Diagnosis.**—The extreme importance of early and correct diagnosis of small-pox should lead the physician to use every care in the anamnesis of the patient. While the diagnosis may be made with certainty almost at a glance after the typical eruption appears, there are cases without history or with misleading history, with atypical rashes and eruption, in which certainty of diagnosis may be impossible. The physician should in such cases take all the usual precautions to prevent the spread of the disease, acting as if it were certainly small-pox, and should then await developments. The mildness of the recent epidemics caused the utmost confusion. In a railroad-grading camp at La Veta Pass, where 1,200 men were employed, the disease was so mild that we quarantined the camp, vaccinated every one, and permitted scores of the men to work with the shovel and scraper while the eruption was in full flush, and without any detriment, so far as I could learn, to a single individual. A similar course was pursued in other camps during the same epidemic, yet the occasional development of cases of confluent or hemorrhagic small-

pox, with fatal results, convinced the most skeptical that there could be no discussion even of the nature of the infection.

In the initial stage the disease may be suspected, but absolute diagnosis is impossible, since every feature may be duplicated in other affections. If exposure be admitted, especially in an extensive epidemic, the occurrence of rigors, vomiting, headache, and intense backache should lead to the suspicion of small-pox, and the isolation of the patient, until the eruption appears or the time for it is passed. The prodromal rashes are neither sufficiently frequent nor sufficiently typical to be of much avail, excepting when the petechial form occurs in the triangle of Simon, covering the lower abdomen, the genital region and the upper and inner surfaces of the thighs, on the second day after the initial symptoms mentioned. The measly rash leads to the diagnosis of measles occasionally, though the absence of catarrhal symptoms, cough and Koplik's spots should ordinarily prevent this error. During an epidemic of variola, the contrary error is more frequent and more excusable. Absence of vaccination scars, with history of exposure, raises a presumption in any given case in favor of small-pox. The presence of sore-throat, and the brilliancy of the rash with its rapid diffusion from the point of origin upon the chest and throat, and the absence of petechiæ, offer such good grounds for the diagnosis of scarlet fever that error is not likely to occur.

With a magnifying glass and a good light, the eruption, especially where the skin is thin, may be seen to be vesicular, when it appears, upon casual examination, to be merely papular. The appearance of vesicles upon the palms and soles is commonly decisive. In the colored race the eruption upon the hard palate and other mucous surfaces is of utmost value in doubtful cases. Since in malignant purpuric small-pox the patient may die before the eruption appears, dependence must be placed upon the history of the exposure, absence of efficient vaccination, characteristics of the initial symptoms and the overwhelming severity of the onset. The shotty papules at the edge of the hair upon the forehead may appear and confirm the diagnosis before death occurs.

**Differential Diagnosis.**—Almost insurmountable difficulties arise

in the differential diagnosis between mild forms of small-pox and varicella, and the physician in such difficulties may comfort himself with the thought that even Hebra believed in the unity of the two diseases. Such a position is now wholly untenable. The seat of the lesion is in the epidermis in both diseases and it is vesicular in both. The sharper, rounder vesicle of varicella contains a clearer fluid, and it may all be evacuated by a single needle prick, being in a single cavity, contrary to the findings in small-pox. The greater rapidity of development in chicken-pox, and the development of the papule upon the hyperemic base, the appearance of successive crops, so that macules, papules and vesicles are present at one and the same time, the absence of true pustules, and of primary umbilication should be noted. Certain vesicles become so cloudy, and the resorption of their contents presents such fairly marked secondary umbilication, that careful examination is necessary.

The absence of the shotty feel found in small-pox, presence of eruption behind the ears, rapid drying up of the vesicles and absence of secondary fever are further points in favor of varicella.

The diagnosis of chicken-pox in the adult should be regarded with suspicion because of its comparative rarity.

Abortive small-pox gives rise to great difficulties in diagnosis, the eruption not progressing far enough to present the decisive features. The patient must be isolated and the usual precautions taken. In view of the mild epidemics of variola of the last dozen years no great weight is to be placed upon the severity of the initial symptoms, place of first occurrence of rash, or exact date of the eruption. The spread of the epidemic in the West was readily accounted for by the occurrence of scores of cases so mild that the patients did not stop work for an hour, unless apprehended late in the course of the disease by the health authorities, after a new focus of infection had been suspected and sought for.

The occurrence of the two diseases at once may cause confusion. Thus in a recent case, an unvaccinated boy of five years, without known exposure to varicella, presented a typical rash of that disease, thirteen days after an undoubted case of small-pox had been removed from the next room in the hotel. The appearance of petechiæ,

with the history, and in spite of the characteristics of the eruption, led four of us, three of whom had had very extensive experience with small-pox in various official capacities, to change the diagnosis for a few hours to that of hemorrhagic small-pox. The further developments showed beyond any question that it was a case of chicken-pox complicated by purpura fulminans, fatal at the end of 20 hours. The latter disease is excessively rare (less than 20 cases recorded), often follows varicella, and the single subcutaneous hemorrhages may be large enough to cover perhaps an eighth of the body surface, as in the case quoted. Death may be even more rapid than in purpura variolosa.

**TYPHUS FEVER.**—This disease may present the sharp onset of variola and a petechial rash, but no vesicles or pustules appear, the rash is not shotty to the feel and the fever does not subside with the eruption.

**SYPHILIS.**—A pustular syphilid may very closely resemble the eruption of small-pox, but there the similarity ends. The patient is likely to be walking about, without fever, and suffering only because of his appearance. The history, adenitis, lack of progress in the eruption, finding of the spirochetes or the positive Wassermann reaction should decide the question promptly.

**CEREBROSPINAL MENINGITIS.**—The malignant form of this disease, with subcutaneous hemorrhage, may closely resemble hemorrhagic small-pox, and diagnosis may be impossible unless spinal puncture enables us to find the meningococcus, or the appearance of the shotty rash settles the question.

**GLANDERS.**—A few cases of pustular eruption in this disease have been reported in America. The history and initial symptoms are unlike those of small-pox. Fetid nasal discharge should arouse suspicion, and the *Bacillus mallei* should be sought for.

During the Civil War prisoners escaped from military prisons by inducing a facial pustular eruption through application of croton oil. The slightest examination of the case would expose such deception. In case of dispute, histological examination of the excised skin lesion in small-pox offers absolute evidence of its identity. Corneal inoculation in the rabbit may also be relied upon.

**Prognosis.**—In unprotected races small-pox is extremely virulent and many tribes and even nations have been practically wiped out by the disease. The mortality before the introduction of vaccination was not infrequently 50 per cent. or more.

Cases of varioloid always recover; discrete small-pox offers an excellent prognosis; the confluent form has a mortality exceeding 50 per cent. *Variola pustulosa hemorrhagica* may recover, while *purpura variolosa* is always fatal.

Epidemics may offer the greatest diversity as to the mortality rate, aside from the protective influence of vaccination. We may speak confidently upon this point because extremely mild epidemics were recorded before Jenner's discovery was made. The mortality of 6,942 cases coming under the supervision of the Colorado State Board of Health during four years of my connection with it was but 20, approximately  $\frac{1}{4}$  of one per cent. Amongst the unvaccinated, mortality rates of 50 per cent. to 70 per cent. have been recorded during the past 30 or 40 years. Welsh and others have shown that the mortality rate from small-pox decreases in almost exact proportion to the efficiency of vaccinal protection, as judged by the number, area, and character of the vaccination scars.

Youth, and especially infancy, alcoholism, debility, pregnancy, the presence of complications, especially in the larynx and lungs, and the general severity of the individual epidemic are features calling for grave prognosis.

The physician suddenly exposed to small-pox long after vaccination or revaccination may best protect himself by immediate vaccination with two or three reliable vaccines obtained from different sources, with the idea of obviating the possibility that age, exposure to heat or other agencies may have rendered any given vaccine inert.

## II. VACCINIA

**Definition.**—Vaccinia or cowpox is of interest to the physician because the transmission of the disease by vaccine to the human subject produces a pock and a general febrile disturbance which are followed by a more or less complete immunity against variola. By

vaccination we mean the introduction of the virus of cowpox, for the establishment of such immunity. Bovine vaccine lymph is now used almost exclusively in this country.

After vaccination there follows a period of incubation of three or four days, and then the local reaction begins as a reddish papule, or several of them, at the site of inoculation. A red areola develops and by the sixth day the vesicle is well formed, distended with clear lymph, somewhat umbilicated. Till the ninth or tenth day the swelling increases, the vesicle grows, the umbilication becomes more marked, the content becomes more purulent. By this time the moderate fever which has accompanied the process disappears, the swelling lessens, the vesicle begins to dry up and form a scab, the local adenitis subsides, and generally during the fourth week the scab becomes detached, leaving a reddish scar which gradually becomes white and pitted (foveated). Secondary vesicles are fairly common near the site of vaccination, and generalized vaccinal eruption may occur, coming in successive crops, and perhaps lasting for weeks. Confluence of several pocks may be seen. The virus may be carried to various places, either immediately after the vaccination or after the vesicle has developed. I have known a typical vaccinal vesicle to appear upon the chin coincident with the development of the one upon the arm, the patient having cut his face while shaving as he waited for the vaccine to dry. In another case a mother scratched her baby's arm after vaccination, infected her own vulva, transmitted the infection to her husband, and thus raised the question of venereal disease, which was, happily, easily disposed of.

For information as to variations in the course of the vaccinal disease the reader is referred to the special works. Acceleration or retardation of development, abortive development, or other anomalies ordinarily call for revaccination, which of course may not "take."

A moderate leukocytosis is noted at the time of development of the vesicle. The possible transmission of tetanus, erysipelas, syphilis, tuberculosis, and possibly other diseases should lead the practitioner to use every care in the selection of the virus and the method of vaccination. A scar of unusual depth and size is likely to result

rather from secondary infection than from the vaccination proper, and may not be evidence of immunity.

Ackland's summary of the eruptions or complications occurring at different dates is valuable:

"1. During the first three days: erythema; urticaria; vesicular and bullous eruptions; invaccinated erysipelas.

"2. After the third day and until the pock reaches maturity: urticaria; lichen urticatus; erythema multiforme; accidental erysipelas.

"3. About the end of the first week, and generally after the maturation of the pocks; generalized vaccinia,—(a) by auto-inoculation, (b) by general infection; impetigo; accidental erysipelas; vaccinal ulceration; glandular abscess; septic infections; gangrene.

"4. After the involution of the pocks: invaccinated diseases, for example, syphilis."

Children should ordinarily be vaccinated in the third month, but earlier if exposed. Revaccination should be done every seven years until puberty is passed and thereafter when exposure demands.

Protection against small-pox is fairly assured by the eighth or ninth day after vaccination if the latter be typical. It is possible to take small-pox, soon after what appears to be absolutely typical vaccination, and on the other hand, I have seen vaccination develop perfectly upon the arm of a nurse vaccinated *after the beginning of the eruption* in a case of small-pox, of such severity that more than 100 pustules appeared upon the soles of the feet. It should be stated that these limitations as to the protective influence of vaccine should only obligate the physician to use greater care in diagnosis and in securing the almost absolute protection against small-pox which early and repeated vaccination affords.

### III. VARICELLA

**Definition.**—Varicella or chicken-pox is an acute infectious disease, chiefly of childhood, endemic and epidemic, presenting an eruption somewhat resembling that of variola, but being nevertheless a distinct disease. Varicella is constantly confused with mild small-

pox, but neither disease gives rise to the other, nor protects against the other. Vaccination has no protective influence against chicken-pox. The direct causative agent is unknown.

The period of incubation is probably 12 to 15 days in most cases and is without symptoms. After an initial chill in the more severe cases, or headache and malaise only in the milder ones, the eruption appears, usually within 24 hours. The temperature, which may have reached 100° F., subsides upon the appearance of the rash unless the latter be unusually abundant. The rash appears first upon the face and neck and spreads so that the body is commonly more affected than the limbs and face, contrary to the distribution of small-pox. The preliminary papule may develop into a full-fledged vesicle in a few hours and most cases present vesicles when examined by the physician.

The vesicles are discrete, often but few in number, superficial, with a little surrounding redness, and contain a clear serum, later becoming cloudy, and completely evacuated by means of a single puncture. The eruption is rarely seen on the palms and soles, but often behind the ears, and upon the buccal surfaces. The vesicles here rupture early and then present slight erosions only. The appearance of the eruption in successive crops is characteristic of chicken-pox, as contrasted with variola. Macules, papules, vesicles, and other vesicles with such cloudy contents that they appear at first sight to be pustules are present together. A secondary umbilication may be noted. After three or four days the vesicles dry up, and the crusts soon become detached. Scars result easily, especially upon the face, if the vesicle be ruptured and secondary infection occur.

The disease may last three weeks or more before the last crusts separate. Permanent immunity is commonly acquired.

The skin lesions may become bullous or hemorrhagic, and gangrene may occur. I have seen death from noma and from purpura fulminans.

**Diagnosis.**—This chiefly concerns the differential diagnosis from small-pox, which has already been considered. Corneal inoculation and histological examination of the skin lesions are decisive.



**Prognosis.**—This is almost invariably favorable, excepting in feeble cachectic children.

#### IV. MEASLES

**Definition.**—Measles (rubeola) is an acute, highly infectious disease occurring in epidemic form, with coryza, bronchitis, and a characteristic eruption. It is especially a disease of childhood, but probably chiefly because adults have nearly all had the disease during that time. In communities exposed for the first time, nearly all acquire the infection, as in the oft-quoted epidemic in the Faroe Islands, where nearly 80 per cent. were infected in the first wave of the invasion.

Measles is the most widely disseminated infectious disease. The active infectious agent has not been demonstrated. The disease is nearly always communicated by direct exposure, often before the appearance of the eruption, but it is probable that fomites may occasionally be responsible for its spread. The infection is certainly less persistent than in most of the infectious diseases, but it is extremely active. Natural immunity to the disease is comparatively rare. The association of measles with whooping-cough and varicella, together or during the same winter, should be noted.

**Symptoms.**—The period of incubation is probably usually nine to eleven days, the eruption appearing upon the thirteenth or fourteenth day. Feverishness and loss of appetite frequently appear at this time. Rolleston found preliminary rashes in over 40 per cent. of the cases, often on the day of the onset of catarrhal symptoms. The prodromal rash may be erythematous, scarlatiniform, or urticarial, and is transient. A moderate leukocytosis and slight loss of weight may be found during the incubation period.

**Invasion.**—Drowsiness is so frequent on the ninth or tenth day as to deserve consideration. Following it the patient complains of headache and malaise; chilliness is common, but chill and convulsions are rare. Shortly the conjunctivæ become injected; coryza, sneezing, cough, and hoarseness appear. The temperature reaches 102°, 103°, or 104° F., falling slightly before the eruption, to rise

still higher at that time, and continue for perhaps a day after. Coated tongue, nausea, and vomiting may be noted. The photophobia is extremely characteristic of measles. The eruption first appears in the mouth, generally as discrete reddish blotches, but little elevated above the surface. This enanthem is soon found on the other mucous surfaces, notably the larynx, and is regarded as the cause of the coryza and other symptoms, which have their points of origin there. The diarrhea so often seen may be accounted for by a similar enanthem.

The exanthem appears generally upon the fourth day, occasionally several days later, first upon the forehead, chin, and face, and spreads rapidly, covering the scalp, neck, and back, then the upper extremities, anterior surface of the trunk and the legs within 24 to 36 hours. Before the definite rash appears, a marbling of the skin may be noted. The reddish spots which constitute the beginning of the rash soon increase in size and number and group themselves into roughly crescentic patches. On the face and upper chest the entire surface may be covered. The eruption darkens in tint and fades after several days, becomes yellowish-brown and the slight pigmentation left disappears in the third week. A small vesicle is occasionally seen. In "black measles," a form not infrequent in certain epidemics, hemorrhage occurs about a majority of the spots and even from the mucous membranes. A few petechiæ, especially about the wrists and ankles are found in perhaps 5 per cent. of the cases of measles, may even be produced by bruising or pressure upon the skin, and are of comparatively little significance.

The rash varies much in different cases. It may appear to be deep in the skin; the spots may be distinctly papular, remotely suggesting the papular stage of small-pox; the rash may show intensely if the patient have good circulation and the skin be warm and moist, as after a hot bath, or may fade almost completely if the contrary conditions obtain (the "striking in" of the laity, regarded by them as fatal). The disappearance of the rash because of cardiac failure is of grave omen.

Measles may occur without fever, without exanthem, or without enanthem. A malignant form, with high fever, intense toxemia,

coma, adynamia, and hemorrhage has occurred more often in peoples unprotected by previous racial experience with the disease. During the height of the eruption the lymph nodes, especially of the neck, are commonly enlarged and tender. After the rash fades a fine branny desquamation occurs, beginning on the face and being complete in about a week. By this time all the symptoms have disappeared unless it be the cough, which occasionally persists after convalescence is fully established.

**KOPLIK'S SPOTS.**—During the stage of invasion Koplik found "on the buccal membrane and inside of the lips . . . a distinct eruption, which consists of small irregular spots of bright red color. In the center of each spot there is noted in strong daylight a minute bluish-white speck." The spots are regarded as pathognomonic of measles, and the exanthem may be confidently expected if they are discovered. They disappear or lose their distinct characteristics before the eruption upon the skin is fully developed.

No definite laboratory findings are associated with the disease. A study of the blood is of comparatively little assistance in diagnosis. A slight leukocytosis occurs early in the incubative period while leukopenia may be present at the time of the eruption. Serious complications may be indicated by the appearance of a polynucleosis. In case of doubt, as between measles and scarlet fever, leukocytosis points toward the latter.

Albuminuria and the diazo-reaction are found in a majority of the cases.

**Complications and Sequelæ.**—The mortality of measles depends rather more on the complications than the original disease, and the well-recognized fatality of epidemics in foundling hospitals with bad hygienic surroundings, etc., depends upon the extension of the catarrhal processes of measles beyond the usual limits. Diarrhea is common during the height of the eruption. Epistaxis may occur.

The most important and most fatal complication is bronchopneumonia, dependent upon the extension of the catarrhal bronchitic process into the finer bronchial tubes and the alveoli. Exposure to cold is a common cause of its development. Lobar pneumonia, pleurisy, empyema, and gangrene of lung are much less frequent. The

almost constant enlargement of the tracheo-bronchial lymph glands is largely responsible for the harrassing cough which may last for months after a severe attack of measles. I have seen suppuration, with rupture of the abscess resulting into the esophagus, and recovery. The frequent incidence of tuberculosis in those recovering from measles may be explained by infection from without, during the period of lowered resistance, or by the outbreak of an acute process in an old tuberculous focus.

Catarrh of the middle ear, otitis media (24 per cent., McCollum) with rupture of the drum and occasionally mastoid involvement, are common. Moderate deafness often results. Conjunctivitis and photophobia are parts of the disease, and ulceration of the cornea may occur.

Nephritis is uncommon. The catarrhal laryngitis of the disease may be complicated with a membranous deposit, either streptococcic or due to the diphtheria bacillus. This complication is very serious. Persistent hoarseness may be a sequel. Gangrenous stomatitis is occasionally seen in feeble children, and milder types of stomatitis are comparatively common. Cardiac complications, aside from exhaustion, are rare. Phlebitis has been noted. A post-febrile toxic neuritis is a rare sequel. Arthritis and osteomyelitis have been reported.

The occurrence of the enanthem may cause so much distress in the abdomen as to simulate acute appendicitis. In a nurse at St. Joseph's Hospital the pain, fever, rigidity, and tenderness led to early operation. The eruption showed upon the peritoneal coat of the exposed bowel and of the catarrhal appendix, which was removed. Within an hour after, the exanthem appeared.

Intestinal catarrh and chronic colitis may occur as sequels of measles.

**Diagnosis.**—This is rarely difficult, for the coryza, bronchitis, Koplik's spots, photophobia, and eruption are characteristic. Before the eruption appears, the coughing, lacrymation, sneezing, and Koplik's spots render the case extremely suspicious, and it should be isolated. For the differential diagnosis the reader is referred to the Sections under Scarlet Fever (page 564), Rotheln (page 558),

and small-pox (page 545). The roseola of syphilis has not the catarrhal symptoms of measles and the patient is not so acutely ill. Typhus and typhoid with facial efflorescence rarely leave us in doubt, for the catarrhal symptoms are absent in the latter, and all symptoms are much more intense in the former, and the eruption is commonly hemorrhagic. Influenza and the rashes after the use of sera and after certain drugs should all receive consideration, but usually offer no great difficulties.

**Prognosis.**—The mortality varies enormously in different epidemics, and especially under different conditions as regards the resisting powers of the patients and the hygienic surroundings. In general, probably a 3 per cent. mortality might be taken as an average in ordinary epidemics under favorable conditions, but in foundling asylums, and barracks, and in soldiers in time of war (as in the Civil War) the rate rises toward 30 per cent. and even 50 per cent. 30 per cent. to 40 per cent. of the cases complicated with bronchopneumonia are fatal, the majority falling in the period of early childhood. The prognosis in hemorrhagic measles is always grave, but less so in case the hemorrhage appears late during the evolution of the eruption. A few petechiæ about the wrists and ankles do not signify especial danger. The outlook becomes worse after middle age. The relative mortality of measles under average conditions may be judged from the report below of six important infectious diseases during the first four years of the time of my official connection with the Colorado State Board of Health:

DISEASE	CASES REPORTED	DEATHS	PERCENTAGE MORTALITY
1. Typhoid .....	6948	1,058	15. +
2. Scarlet fever .....	9260	618	6.6
3. Diphtheria .....	5388	887	16. +
4. Small-pox .....	6942	20	.28
5. Measles .....	(not reportable)	178	....
6. Whooping-cough .....	(not reportable)	162	....

## V. RUBELLA

**Definition.**—Rubella (rotheln: German measles) is an acute infectious epidemic disease, characterized by enlargement of the post cervical glands, a cutaneous eruption, and by the mildness of the constitutional symptoms. In different forms it resembles both measles and scarlet fever, but it is different from either.

Although the disease is readily transmissible and occurs in extensive epidemics, its active contagious principle is unknown. The infective period begins before the appearance of the eruption, and continues well into convalescence. Second attacks may occur in two or three per cent. of the cases. Children are chiefly attacked, but adults are by no means immune. The sexes are equally liable to the infection.

**Incubation.**—The period is thought to be generally from ten to fifteen days, though occasionally much shorter, and extending in other cases to twenty days or more. Prodromal symptoms are those of slight malaise, if any appear, with occasional reddening of the eyes, and sneezing.

**Invasion.**—This lasts from a few hours to forty-eight hours. The patient complains of malaise, coryza, pharyngitis, and slight sore-throat. A rise of temperature of one to three degrees is present for a day or two. The rash may be the first sign to attract the patient's attention. It commonly appears upon the first day, but may come on the second or third. It affects the face and scalp first, and spreads downward. It consists of roundish or oval measles-like spots, without the crescentic arrangement of measles. On certain parts of the body the rash is frequently indistinguishable from that of scarlet fever, notably where pressure occurs, and on the buttocks and inner aspects of the thighs. The spots may be slightly papular. The rash may appear in successive crops. The eruption remains out 2 to 5 days and may be disappearing in the parts first affected before it has completely developed in other regions. Slight desquamation follows.

Forchheimer described a macular, rose-red eruption upon the palate and uvula coincident with the exanthem.

Congestion of the eyes and slight swelling of the pharynx and tonsils is commonly present. The most distinctive feature of the disease is the swelling of the post-cervical glands in particular, though others are often slightly involved. This may show before the eruption, and resolves shortly after its disappearance. The swelling may be seen as well as felt in marked cases. Suppuration probably never occurs. The blood findings are of little importance and resemble those of measles. Complications are unusual, but may occur along the same lines as in measles. There are no important sequelæ. Relapse is rare.

**Diagnosis.**—This is easy in epidemics, being based upon the lack of constitutional symptoms, the polymorphous rash, the eruption upon the palate, glandular enlargement, the lack of throat symptoms of scarlet fever, and the catarrhal manifestations of measles. Because of the epidemic character of rotheln, doubtful sporadic cases are more apt to turn out to be instances of scarlet fever or measles. In the mildest forms of these two diseases confusion may exist, but the vomiting at onset, characteristic tongue, severity of desquamation, and frequency of post-scarlatinal nephritis offer further points of distinction as to the former, and the crescentic arrangement of rash, Koplik's spots, and different distribution of the glandular involvement, as to the latter. The "fourth disease" of Dukes has not been recognized as sufficiently distinctive to entitle it to recognition, but resembles Rotheln more closely than measles or scarlet fever.

The erythema infectiosum of Escherich is a feebly contagious disease of children,—not developing in America, or not recognized,—with a rose-red maculopapular rash and without constitutional symptoms or mortality.

**Prognosis.**—Recovery occurs in rubella unless some unusual complication brings about a fatal result. No death has been officially reported in Colorado during many years.

## VI. SCARLET FEVER: SCARLATINA

**Definition.**—Scarlet fever is an acute febrile disease, highly infectious, characterized by angina, and a scarlet rash, and followed by desquamation.

It is a disease of cities rather than the country, of tenement houses rather than separate dwellings, and of cold weather rather than warm, greater exposure being the essential factor in all. It differs from small-pox, chicken-pox, and measles in that many individuals and often whole families are immune, and especially after the age of puberty. It is quite possible that mild unrecognized attacks in childhood are the real basis of this immunity.

The infectious agent has not yet been positively identified. Streptococci are found in the throat during the disease, but apparently are not the cause of the disease. Mallory's protozoan may yet prove to be the causative organism. The disease is intensely infectious, and the infection is easily carried by the hair, clothing, bedding, toys, cats, birds, etc. Clothing folded away for years even may transmit the disease. It has been carried in the milk in some epidemics, often of a very virulent character.

The period of incubation is believed to be short in many instances, often not over two to five days, but may be much longer. McCollum, who has had an unrivaled experience, believes it is generally over six days, and often ten to twenty.

**Symptoms.**—The disease commonly begins with vomiting, chill being comparatively rare. Convulsions are common in young children. A sharp rise in temperature to 104° or over occurs in the next few hours, and the throat becomes reddened and sore. Within a period varying from two or three to twenty-four hours after the vomiting the rash appears, most frequently in twelve hours, more or less. Appearing upon the neck and chest, it spreads over the entire body within the next twenty-four hours, the face often escaping. At first definitely punctate and often remaining so upon the legs, the skin between the points becomes reddened and slightly swollen, so that in a well-marked case, the whole body appears scarlet. The rash is really erythematous in character, disappearing upon pressure, but instantly reappearing. It may cover the parts first involved, is often absent about the mouth, may be "patchy," may be modified by the presence of sudamina, and petechiæ may appear in severe cases. In malignant types extensive ecchymoses may appear.

The throat and roof of the mouth are intensely red. The



tongue is commonly coated white, with the red papillæ showing through (strawberry tongue). The papillæ are always enlarged and after the coating has disappeared they show very distinctly. At this time they may be of great value in diagnosis. The rash persists for from two to four days and then fades. Desquamation succeeds it shortly, being delayed in the milder cases to ten or even twenty days from the beginning, and being then of a mild type. Desquamation may even be absent in the mildest cases. In those patients with brilliant rash and high fever, the scales may separate as lamellæ and the skin of the hands and feet may come off like parts of a glove. Desquamation is commonly complete in five or six weeks, but may last a fortnight longer. While desquamation is often the best evidence of a pre-existing scarlet fever, and, for example, in the face of an unexpected acute nephritis, is trustworthy evidence, it occurs so often after almost any severe inflammation of the skin that it alone should not be relied upon as evidence that the patient has had the disease.

The tonsils often present the appearance of an ordinary follicular tonsilitis, but not infrequently a membrane indistinguishable from that of diphtheria is present, and the Klebs-Löffler bacillus is not infrequently found. Intense inflammation, edema, and swelling of the tissues of the neck occur, cervical adenitis is marked (fifty per cent.) and suppuration of the glands of the neck is present in eight per cent. of the cases (McCollum).

The temperature rises in the average case to 103° or 104° F., and continues high with slight remissions until the rash begins to fade, and then falls gradually, to disappear in normal cases at the end of the first week or soon after. Hyperpyrexia occurs in certain severe cases, and low temperature is not infrequent. The fever may disappear by lysis or by crisis.

The pulse is rapid, often reaching 150 per minute and is commonly full and tense. A leukocytosis is present (18,000 to 40,000) during the height of the disease, falling to normal with recovery. A slight secondary anemia is the rule in severe cases. The examination of the blood is of little value clinically in this disease.

The spleen is slightly enlarged and may be palpable. A febrile

albuminuria exists in nearly three-fourths of the cases, disappearing with convalescence. Acute nephritis may occur later.

Headache, insomnia, and delirium are common features in severe cases.

**Types.**—Scarlet fever is commonly divided according to its severity into the following types:

(a) **FULMINATING** (*Scarlatina Siderans*).—The onset is of great severity, convulsions, chill, vomiting, diarrhea, headache, delirium, coma, and high fever being common. I have seen death in eighteen hours, before any rash occurred, the diagnosis being based primarily upon the presence of the disease in the family.

If the child lives twenty-four to forty-eight hours a dark-red rash may appear. The angina may be inconspicuous. Failure of the pulse and suppression of urine are noted toward the end. The case may be mistaken for one of malignant small-pox.

(b) **HEMORRHAGIC FORM.**—This is a severe type, accompanied by petechial hemorrhages, often very extensive, and by bleeding from the nose and the kidneys. It is commonly fatal in two or three days. It is rare unless in the feeble inmates of foundling asylums, etc.

(c) **ANGINOSE FORM** (*Scarlatina Sine Eruptione*).—In this variety the throat symptoms overshadow the eruptive manifestations. The tonsils, pharynx, fauces, and nasal cavities are intensely inflamed, and commonly covered with a false membrane, often streptococcic in origin. The diphtheria bacillus may be present. Extensive sloughing, foul, purulent discharge from the nares, extension through the Eustachian tube and the ear, severe adenitis, general sepsis, hemorrhage at the time of separation of the sloughs, dysphagia, obstruction of the air passages, complicating pneumonia, cyanosis, stupor, and death are frequent manifestations of the severity of the infection of this type.

(d) **ORDINARY FORM.**—This has already been described as the type of the disease.

(e) **MILD FORMS.**—The disease is often so mild that the faint, transient, limited rash is not noted by the mother, if it appears at all. The patient continues with his play, and the diagnosis is not made until other cases appear or desquamation and nephritis call

attention to the matter. This type is responsible in great measure for the spread of the disease. Another mild form may be introduced by vomiting and slight angina, but no rash is found, or only a faint flush. In similar manner the angina may be absent in certain of the milder cases.

A protracted severe form of scarlet fever is mentioned by some writers, with a high fever, delirium, and profound prostration, which signify a severe infection. Surgical scarlet fever is a misnomer, the rash in septic surgical cases being due to the streptococcus in most cases. Puerperal sepsis may develop from exposure to scarlet fever, probably from the streptococcic throat infection. Surgical or other patients may of course contract scarlet fever in the usual forms.

Quinin and other drugs may be taken by a patient with tonsillitis and may produce an erythematous rash. The deception may be complete until the rash disappears the next day, as I know by experience. Mild desquamation may follow.

Scarlet fever varies more widely in the different types than do the other infectious fevers, and probably more cases fail of recognition because of the mildness of many of them than in any other serious disease.

**Complications and Sequelæ.**—KIDNEYS.—Acute nephritis is the most common serious complication. It occurs most frequently in the third week and more often proportionately in cases not recognized, and therefore not properly cared for. Its appearance seems to bear no especial relationship to the febrile albuminuria seen in most cases early in the disease. There may be any grade of infection from a mild catarrhal nephritis to a severe and rapidly fatal glomerulonephritis. In the milder cases albumin and a moderate number of hyaline and granular casts, with perhaps epithelial casts and epithelial cells, but no free blood, are found. In severer types albumin is more abundant, the urine is scanty and darker colored, often smoky from destruction of the red-blood cells, and epithelial and blood casts and free epithelial and blood cells are abundant. Edema or general anasarca, suppression of urine, convulsions and death may follow. In intermediate types the symptoms are less severe than those just described, but moderate edema persists, anemia

develops, and the nephritis becomes chronic. Even if recovery ensue, the patient may easily develop albuminuria in later life, after slight exposure. But twenty cases of chronic nephritis developed in McCollum's 5,000 cases.

Edema may be present without albuminuria, presumably from anemia. Its occurrence should call for repeated examinations of the urine.

**HEART.**—Endocarditis or pericarditis may occur in perhaps one or two per cent. of the cases, generally from the associated streptococcic infection. The heart may dilate rapidly with fatal results. The common mitral systolic murmur (twenty to forty per cent.) in scarlet fever is a relative one, due to myocardial weakening, from the toxins of the disease, or to the nephritis.

**JOINTS.**—The so-called scarlatinal rheumatism is better designated as a septic arthritis. It is painful and distressing when affecting several of the smaller joints, but commonly recovers without suppuration or surgical intervention. This variety is presumably due to the toxins of the disease. The streptococcus has been recovered from the effusion which rarely occurs in a single large joint, and these cases may go on to suppuration.

**LYMPHATIC GLANDS.**—The ordinary involvement of the cervical glands subsides without serious symptoms, even though they may be much enlarged and quite tender. The enormous enlargement seen in severe anginose forms of scarlet fever frequently goes on to suppuration and necrosis, perhaps with fatal hemorrhage. Many cases of angina ludovici are merely late sequels of scarlet fever, perhaps six weeks after the beginning of the attack.

**THROAT.**—The throat involvements have been discussed under the anginose form of scarlet fever.

**EARS.**—Infection of the middle ear, most often bilateral, has been mentioned as occurring in the anginose type of scarlet fever, and is to be expected (eighteen per cent., McCollum). Partial deafness from this accident is extremely common, and more than ten per cent. of deaf-mutism is chargeable to it. Perforation of the drum occurs, and frequently extension to the labyrinth, and to the mastoid cells. Necrosis of the temporal bone, meningitis, brain abscess and sinus

thrombosis may occur. Chronic otitis media follows in a large proportion of cases not fatal from other cause.

Amongst other complications which may be seen are pneumonia, bronchopneumonia, pleurisy, empyema, peritonitis, jaundice, furunculosis, paronychia, cancrum oris, venous thrombosis, post-febrile psychosis, etc. Many of these are associated with the streptococcic infection so common in the throat, rather than with the scarlet fever directly. The development of toxic multiple neuritis and vagus neuritis I have been disposed to attribute, in severe cases, to a complicating but undiscovered diphtheria, first, because of the recognized frequency of this complicating disease, and, secondly, because of the frequency of the neuritides as sequels of it.

An attack of scarlet fever commonly confers immunity for life. Doubtless most of the instances of alleged recurrence have been due to the mistake of diagnosing as scarlet fever certain cases of German measles, erythema, septic rashes, drug eruptions, etc. A true relapse may occur four or five weeks after the eruption, and a second infection may occur still later.

**Diagnosis.**—In severe typical cases the diagnosis is made with ease and certainty after the eruption appears. In mild cases, before the exanthem, it is often impossible. The sudden onset, sore throat, vomiting, high fever, nervous symptoms, characteristic tongue, and history of exposure often justify a diagnosis before the eruption appears. When the latter develops, little question commonly exists. In doubt one must isolate the patient and wait until the time for desquamation has passed, though the absence of desquamation is not proof that the patient has not had a mild attack.

From measles, scarlet fever should be distinguished through the presence of catarrhal symptoms, cough, and Koplik's spots in the former. Leukocytosis is absent in measles, the rash is later, different in color and form and less brilliant, and sore throat is absent, or less prominent than in scarlet fever.

**ROTHELN.**—The rash causes confusion, for in certain cases the resemblance is very close. The history of an epidemic, general mildness of the symptoms, presence of post-cervical adenitis, and the absence of angina are usually sufficient for the differentiation. It is

a grave error to diagnose rotheln if any doubt can possibly exist in these cases, and it is better to isolate the child and wait for developments, unless the diagnosis of rotheln is well founded.

**DIPHTHERIA.**—If this disease exists alone and without a complicating septic rash no cause for confusion exists. If the rash be present, the finding of the Klebs-Löffler bacillus does not exclude scarlet fever. The occurrence of nephritis as a sequel points toward scarlet fever, while involvement of the vagus or its branches, or multiple neuritis, should incline us toward a diagnosis of diphtheria.

Acute exfoliative dermatitis, septic rashes, drug eruptions, erythemas due to food infection, and the rashes of various acute diseases must be considered in certain cases, but in none of these are there constantly present, as in scarlet fever, an acute onset, angina, and typical rash at the proper

interval. In cases of doubt, and they are not rare, isolation should be the rule until the doubt is resolved.

**Prognosis.**—From four to fifteen or eighteen per cent. of cases, if a large number be studied, are fatal. The mortality varies greatly in different epidemics. The disease is much more fatal in babies than after the sixth year, and becomes less severe after the twelfth year. The ordinary and mild cases usually recover, the anginose



FIG. 35.—DRUG ERUPTION. Balsam of copaiba.  
(From the collection of Dr. A. J. Markley.)

cases are severe, and the hemorrhagic ones generally fatal. Severe complications render the outlook grave. No case can be pronounced out of danger until the time for the occurrence of acute nephritis has passed. Of 9,260 cases reported to the Colorado State Board of Health in 1901-4, 618 died, a mortality rate of approximately 6.6 per cent.

## VII. MUMPS

**Definition.**—Mumps (epidemic parotitis) is an acute infectious disease characterized by inflammation of the salivary glands, especially the parotids, and with frequent metastases to other organs. It is often epidemic in cities, and endemics are frequent in schools and other institutions where the young are gathered together, young adults, as in barracks, and the higher schools, often suffering. Many individuals are naturally immune, and of those attacked many suffer upon one side of the face only. More boys than girls are affected. The exact cause and method of infection have not been demonstrated, although in most cases certainly personal contact with a previous case may be assumed.

**Symptoms.**—The incubation period is probably two or three weeks. Then the patient may suffer from malaise, headache, nausea, and occasionally vomiting, although many patients notice nothing until the swelling of the parotid occurs, more commonly the left. The gland enlarges rapidly for two or three days, with a fever of  $103^{\circ}$  in the average case, difficulty in opening the mouth, in speaking, swallowing, and in mastication. The gland becomes much swollen, tender to the touch, with a tense edema over it. The skin may be blanched by the swelling and tension. A pushing outward of the lobule of the ear, with widening of the face, is characteristic. Fetid breath, earache, slight deafness, tinnitus, and otitis media may be noted. The other gland is likely to become involved in a day or two, and other salivary glands may participate.

A feeling of tension and pain is present. Sharp pain may be felt in the affected side upon tasting a lemon, pickle, etc.—a diagnostic point among the laity. Pick found a slight relative increase in the mononuclear cells only.

**Complications.**—Suppuration of the parotid gland occurs only from a mixed secondary infection. I have known of it but once in epidemic parotitis. The gland may remain permanently enlarged in other cases. Orchitis occurs in practically a third of the males, generally a few days later than the parotitis, and more often of but one testicle. In a small epidemic under my observation it occurred in every one of the thirteen males affected. The epididymis is often involved, and the swelling may be enormous. A non-specific urethritis has been seen. Subsequent atrophy of the testis often occurs, and if double may involve loss of procreative function. Ovaritis occurs in females past the age of puberty, but it is infrequent. It may cause sterility. Mastitis may be noted in either sex. The involvement of the testis or ovary may be the sole manifestation of the disease. Other complications are infrequent, deafness being perhaps the most common. Pancreatitis has been observed.

The febrile symptoms disappear after six or eight days, but the swelling of the parotid, testicle, etc., often persists for a week or two after.

**Diagnosis.**—This is generally easy. The location of the swelling in front of and below the ear, pushing the lobule outward, the widening of the face, absence of palpable adenitis, and of inflammatory infections of the throat, and prompt resolution should suffice.

The parotitis of the infectious fevers, notably of sepsis and typhoid, commonly suppurates, is rare in children, and is the sequel of a serious febrile affection. The not infrequent incidence of parotitis after abdominal operations and injuries, and occasionally after other operations, even in the absence of recognizable infections, must be recognized. Cervical adenitis from infection or from leukemia, pseudoleukemia, etc., is easily distinguished. The gaseous tumor, crepitating upon pressure, seen generally below the ear, and capable of filling by blowing, as in a wind instrument, and of emptying by local pressure, offers no difficulties. The same remark applies to local abscesses. Enlargement of the parotid may occur in syphilis, in diabetes, in the insane, in poisoning by mercury, lead, and the iodids, in association with facial paralysis, and in connection with the enlargement of other salivary and lachrymal glands in Mikulicz's



disease. In event of orchitis without parotid involvement, diagnosis would be almost impossible unless gonorrheal or other cause for the orchitis could be definitely excluded.

**Prognosis.**—This is almost always favorable. The possibility of sterility following metastasis should be noted. No death from mumps was reported to the Colorado State Board of Health from 1901 to 1904.

### VIII. YELLOW FEVER

**Definition.**—This is an acute specific infectious disease, characterized by fever in one or often two paroxysms, transmitted by the mosquito, but of unknown infectious principle. The *Bacillus of Sana-relli* has not been accepted as the cause, being regarded as a secondary invader.

Reed, Carroll, Lazear, and Agramonte convinced the scientific world that the *stegomyia calopus* transmits the disease, and that so far no other mode of transmission has been established, excepting the artificial one of injecting the blood of a yellow fever patient into one not immune. The mosquito generally becomes infective twelve days after biting an infected patient during the first three days of his disease, and remains infective for life. The mosquito has been found as far north as 40° and as far south as the Rio de la Plata. It is prevalent in all yellow fever countries.

White races are more susceptible than others, and those unacclimated are extremely susceptible to the disease. One attack confers immunity. It is thought that the immunity of the inhabitants of many infected areas is due to their having experienced a mild attack in childhood. Frost stops the spread of an epidemic by killing the mosquito, although the exact method of its action was unrecognized for a century after the fact was well known.

**Symptoms.**—The period of incubation is from two to six days, and possibly longer in certain cases. No very definite symptoms develop during this period.

The onset is stated to be sudden, often with chill or chilliness, a temperature rising even to 106° F. in severe cases, headache and intense aching in the back and limbs. Furred tongue, photophobia,

sore-throat, flushed face, injected conjunctivæ, with a distinct icteroid hue, nausea, vomiting, gastric pain, burning and tenderness, and constipation are noted. Delirium may succeed the early restlessness. The eyelids and lips are often swollen.

The fever attains its height during the first day, remains somewhat elevated for a day or two longer, and then commonly declines by lysis in favorable cases, to rise again in the majority of instances, as the so-called "secondary fever."

The remission may lead to immediate convalescence and recovery, although the patient is at first prostrated and jaundiced. After two or three days, during which the distressing symptoms of onset have been conspicuously absent, the secondary fever begins. At this time the slow pulse characteristic of yellow fever is to be noted. In the face of continued or even rising temperature the pulse rate drops to 70, 50 or even 30, while the thermometer registers 102–105° F. Although much less striking in the milder cases, in the severe forms this phenomenon is so constant that much dependence is placed upon it by physicians in the yellow fever regions.

Albuminuria generally develops by the third day, increasing with the severity of the disease. Hyaline, granular and epithelial casts now appear, all stained by bile, with blood casts and free blood cells in the severe hemorrhagic cases. Suppression with the convulsions and coma of uremia are frequent in severe cases. Delirium may be present, but many severe cases show no great mental disturbance. The stage of collapse succeeds the intermission, with increased jaundice, pinched face, cold extremities, dull expression, dry brown tongue, sordes, petechial hemorrhages, epistaxis, bleeding from the gums, black vomit, tarry stools and death in many cases. The secondary fever lasts two or three days in favorable cases and subsides by lysis. In severe types it may continue to rise until death occurs.

Carroll states that the leukocyte count remains practically normal or that a slight polynucleosis may occur. The finding of the blood reaction in the vomitus may be of value in diagnosis, before macroscopic hemorrhage occurs.

All having experience with the disease insist upon the enormous variability in the severity of the disease, the milder cases perhaps

not taking to bed, while the malignant type causes death in forty-eight hours. The average duration of yellow fever may be stated roughly as one week.

**Diagnosis.**—The direct diagnosis depends upon observation of the signs and symptoms mentioned, generally in epidemic form and in the regions subject to the disease. The difficulties in the way of correct diagnosis, as in the case of plague in San Francisco, are oftentimes not so much medical as commercial and political.

The differential diagnosis from dengue has been considered. The microscope should eliminate malarial fever in most cases. In the absence of laboratory proof, the presence of albuminuria, black vomit, tarry stools, jaundice, and characteristic facies must be relied upon. The mild cases seen in regions where many of the population are at least partially immune, are difficult of diagnosis even to the expert.

**Prognosis.**—The mortality varies widely in different epidemics. The outlook is especially unfavorable in unacclimated persons, the aged, the alcoholic, and debilitated. Black vomit and uremia are of extremely grave omen. The death rate has varied between ten or fifteen per cent. and eighty per cent in different epidemics.

## IX. DENGUE

**Definition.**—Breakbone fever is a widespread infectious disease of tropical and semitropical regions, with a febrile paroxysm, recurring after a few days. The intensity of the pains in the muscles and joints gives rise to the popular name of the disease. A characteristic eruption occurs. When prevalent it attacks nearly as great a proportion of those exposed as does influenza. Its occurrence in warm regions, the known disappearance when it invades the North in the summer, as in Philadelphia during the revolution, upon the appearance of frost, and its resemblance in certain respects to yellow fever, as regards its spread, led Ashburn and Craig to think it a mosquito-borne disease (*Culex fatigans*). No organism has been demonstrated in the blood, possibly because it is too small.

**Symptoms.**—After an incubation period of three to five days or less in some cases, the disease begins suddenly, with headache, chilli-

ness, and the characteristic atrocious joint and muscle pains. The joints become tender, red, and swollen, as in acute articular rheumatism, and movement is painful. Suffusion of the face and neck, swelling of the cervical glands, nausea and vomiting, soreness of the muscles of the eye upon attempted motion, conjunctivitis, prostration, and delirium follow. A temperature of 107° F. and a pulse of 140 may be attained in 24 hours. Hyperesthesia of the skin, with an erythematous macular or papular rash, are commonly present.

After an average duration of three days the fever suddenly ends, frequently with sweating and diarrhea. The eruption disappears and the patient is fairly comfortable. After two or three days a second milder period of fever occurs, followed by defervescence and slow recovery, often with persistence of the joint and muscle pains. During this relapse the eruption is said to be more likely to be papular or urticarial than during the first attack. Itching is often severe and vesicles may develop. Desquamation may follow. Albuminuria occasionally develops. There is leucopenia, often associated with a relative increase in the number of small leukocytes. Complications are infrequent.

**DIFFERENTIAL DIAGNOSIS.**—In the midst of an epidemic, with perhaps half the population affected, there is little call for differential diagnosis. Influenza resembles dengue in its rapid spread and the great number affected, but commonly has marked catarrhal symptoms, and an absence of eruption, joint pains, and remission. Pfeiffer's bacillus may be found. Acute articular rheumatism has similar joint symptoms, and scarlet fever and measles have eruptions more or less resembling those of dengue, but in other ways no resemblance is to be noted. The chief difficulty is in the diagnosis from yellow fever, since the two diseases are often found side by side. The flushed icteric face, injected conjunctiva, jaundice, black vomit, albuminuria, slow pulse, and high temperature of yellow fever are characteristic. The failure of an epidemic to increase the average death rate excludes yellow fever, as has been noted in Texas. Yet disagreement occurs amongst those thoroughly familiar with both diseases.

**Prognosis.**—Death occurs only in rare cases, presumably because

of some depressing concomitant disease or some serious and unusual complication.

#### X. TYPHUS FEVER: SHIP FEVER, CAMP OR JAIL FEVER

**Definition.**—This is an acute infectious disease occurring in epidemics, especially associated with overcrowding and general lack of hygienic surroundings, beginning suddenly and ending by crisis, after 12 or 14 days, and characterized by an eruption which is commonly petechial, and by delirium and other nervous manifestations similar to but more marked than those of typhoid.

It has long been considered extinct in America save for the limited epidemics in newly arrived immigrants. Brill, Anderson, Goldberger, and others have established the identity of the so-called "Brill's disease," seen chiefly in Russians in New York City, with the mild type of sporadic typhus, and Ricketts has shown that the tabardillo of Mexico is also identical.

**Etiology.**—The *United States Health Reports* state that "it seems likely that the body-louse is probably the usual distributing agent by which the disease is carried from individual to individual. Observation seems to show almost beyond doubt that typhus fever is not spread by fomites or by direct contact with the patients suffering from the disease unless the contact is of such a kind that exchange of body lice become possible."

The infecting organism has not been demonstrated. Excepting in the mild sporadic form the disease has been regarded as highly contagious, but chiefly amongst the poor, ill-fed and ill-housed, and notably in immigrant ships or other confined quarters as the various names applied to the disease would indicate. Attendants take the disease with great readiness, a fact easily accounted for in the new light as to the means of contagion.

The immunity conferred by one attack is probably greater than in the case of typhoid. There is no characteristic pathology.

**Symptoms.**—The period of incubation is probably ten or twelve days. Prodromata similar to those in typhoid often occur. The onset is sudden, generally with a chill or recurring chills, and often

with convulsions in children. Headache, flushed face, mental dullness, and injected conjunctivæ are notable features. The fever rises more abruptly than in typhoid to a maximum of  $103^{\circ}$  to  $106^{\circ}$  or even higher in the first three or four days, remaining elevated, with remissions, until it falls, generally by crisis, on the twelfth to the fourteenth day. Delirium is earlier and more marked than in typhoid and subsultus, coma vigil, and coma may occur.

On or about the fourth day the eruption appears. It consists of irregular rosespots, closely resembling the eruption of measles but associated with a mottled appearance beneath the skin (subcuticular rash) and with definite petechiæ, uninfluenced by pressure, and commonly coming later, often in the fading roseola. The rash often disappears without desquamation by the time of subsidence of the fever.

The coated tongue, later crusted and fissured, constipation, more or less characteristic odor, sordes, cough, albuminuria, retention of urine or loss of control of the sphincters are features of the severer types of typhus. In typhus siderans the malignant course ends fatally in a few days. In ordinary severe cases the fatal result occurs in the second or third week in much the same way as in typhoid, if we except the intestinal complications of the latter disease.

If we add bronchopneumonia to the list of complications of typhoid fever we shall have a complete picture of those of typhus. As in the former disease, complications are frequent, severe and often the direct cause of death.

The New York cases have practically all been mild, with a very low death rate so that they were long unrecognized.

**Differential Diagnosis.**—That from typhoid fever has already been considered. In Ireland during the great epidemics, relapsing fever was generally present with typhus. The relapse after five to seven days of fever and a remission is very striking. The finding in the blood of Obermeier's spirochæta is decisive. Measles in children may present a similar rash upon the fourth day, but rarely hemorrhagic. The cough and coryza are more marked. Plague, malaria and epidemic meningitis may require consideration. The assistance of the bacteriologist is often necessary and absolutely decisive.

Hemorrhagic variola has much more extensive bleeding than we see in typhus, and often internally as well as externally.

**Prognosis and Mortality.**—The average death rate is from 10 per cent. to 20 per cent. much less in children, and greater beyond middle age. Pneumonia and bronchopneumonia are often the direct agents in the fatal outcome.

## XI. RHEUMATIC FEVER

**Definition.**—Rheumatic fever (acute articular rheumatism) is an acute infectious disease, of unknown origin, characterized by inflammation of the joints and oftentimes of the endocardium and pericardium.

No bacterial cause is yet commonly recognized. A family predisposition is not unusual. Exposure to cold and wet, and in general, life in a harsh climate predispose to it. It occurs in almost epidemic form at times. Young adults are chiefly affected, and more males than females. No immunity is acquired from an attack, but rather the contrary, for repeated attacks are common.

**Symptoms.**—Prodromes occasionally occur, malaise and sore-throat being most common. A well-defined tonsillitis is not unusual. No definite incubation period is known.

The onset is often abrupt, with chilliness or chill, but is often insidious in children. Fever of moderate degree follows and in the course of a day or two, the joint symptoms appear. One joint or more, frequently several, becomes reddened, swollen and painful, the pain exaggerated by movement.

The tongue is coated and generally moist. The pulse is soft, full and frequent. There are also to be noted loss of appetite, constipation, acid-smelling perspiration and scanty, reddish, acid urine, commonly loaded with urates, and often containing a trace of albumin. With the sweating there is often an eruption of sudamina—at times extremely profuse. Leukocytosis is commonly present. Insomnia may be noted, accounted for in a large degree by the pain in the joints and the general restlessness, anxiety and helplessness. The mind remains clear in most cases.

**JOINTS.**—Almost all the joints may be involved, although commonly the wrists, ankles, elbows or knees are likely to be first affected. Symmetrical distribution is a striking feature of acute articular rheumatism when contrasted with other joint affections. The involvement changes from day to day, certain joints improving and new ones being involved. The small joints of the hands and fingers are less frequently affected. The joints of the spine and the sternoclavicular articulation are most rarely implicated, but no joints are exempt.

The fever varies with the amount of articular involvement, is generally of moderate grade, follows no typical course, declines generally by lysis, and may last from a week to a month or two. Hyperpyrexia is not rare, the fever rising from 104° F. to 108° F., with delirium and stupor, and frequently death in severe cases. Such attacks are more common in the second week. They are rare in America.

Accumulation of synovial fluid is frequently demonstrated in the knees and other joints. I have seen spontaneous dislocation of the hip as the result of such effusion, but it is rare. Subcutaneous rheumatic nodules may appear, especially in children, along the lines of the tendinous insertions. These nodules are especially frequent in cases of rheumatism with severe cardiac involvement. The alleged *diplococcus rheumaticus* may be demonstrated in the nodule, and this fact adds to the probability that the *diplococcus* may turn out to be the cause of the disease. The nodules are really inflammatory exudations, and disappear by absorption after recovery. Erythema nodosum and urticaria may occur. Purpuric hemorrhage (wholly apart from the purpuric manifestation of purpura rheumatica) may occur, especially in the legs.

The joints commonly regain their function completely with recovery. Suppuration is unknown excepting as the result of a secondary infection.

**HEART.**—The most important features of acute articular rheumatism relate to the cardiac involvement. Nearly one half the cases, and a far higher proportion in small children, have endocarditis or pericarditis or both. The heart muscle is often involved.



With the inflammatory affection of the membranes, granular and fatty degeneration of the muscle fibers is often present.

The mitral valve is most frequently involved by the endocarditic process, sclerotic changes being started which eventually cause regurgitation, or less commonly stenosis. The aortic valve is affected only about one-fourth as often, and the two together with about the same frequency. The tricuspid valve in children is occasionally affected, but the right-sided valves commonly escape in adults. Malignant endocarditis is rare in rheumatism. The frequency and severity of the cardiac involvement in children, even in the absence of severe articular manifestations, render acute rheumatism one of the most dreaded diseases of childhood.

Pericarditis is less common, generally fibrinous or serofibrinous, occasionally purulent in children. The pericardial friction sound often masks a soft endocardial murmur. The advent of adhesive pericarditis in children is the most serious common feature of rheumatism, since it is found in the severe types of infection, is accompanied by myocardial changes, and prevents efficient compensation of valvular defects in after years.

Audible and palpable friction, precordial distress, increased frequency of the pulse and respiration, anxious countenance and delirium may be noted. The increase of cardiac area may be as much from dilatation of a heart weakened by accompanying myocardial changes as from effusion. Delirium, lividity and signs of heart failure are of the gravest significance. Chronic adhesive pericarditis, perhaps with mediastinitis and even multiple serositis, may develop years afterwards. In the rare purulent pericarditis the friction may not be perceptible because of the lubricating effect of the pus, but the general signs of extreme danger are present nevertheless.

**LUNGS AND PLEURAE.**—Pleurisy with serous effusion is not uncommon and is to some extent amenable to rheumatic treatment. Pneumonia may occur. In case of heart failure in pericarditis and myocarditis, acute pulmonary congestion may add to the danger. A rheumatic involvement of the peritoneum is recognized by some writers.

**NERVOUS SYSTEM.**—The severer attacks of hyperpyrexia alluded

to are called "cerebral rheumatism" by some authors. The delirium produced by salicylates should be differentiated. The frequent association of chorea with rheumatism has long been noted. It follows the disease in about 15 per cent. of the cases and a micro-organism has been isolated from the brain lesions of a fatal case which produced rheumatic symptoms in rabbits. The severity of the heart lesions in children, in whom the two diseases have occurred together, is very striking. Local neuritis, ordinary multiple neuritis and meningitis may occur. A rheumatic paraplegia has been described.

Phlebitis is a rare complication. Post-febrile anemia is common, and was even more so when the alkaline treatment was in vogue.

Subacute rheumatism resembles the acute form excepting in the milder course and oftentimes longer duration. Chronic rheumatism may result.

**Diagnosis.**—The direct diagnosis is easy in typical cases, and is commonly made by the laity before the physician is called. The frequent history of previous attacks and of tonsillitis, irregular fever of sudden onset, with joint involvement, as outlined above, with pain, sweats, and cardiac involvement, suffice for the diagnosis.

**DIFFERENTIAL DIAGNOSIS.**—This should consider the differentiation from those types of arthritis which occur in connection with various infectious diseases, notably gonorrhea, scarlet fever, sepsis, influenza, meningitis, dysentery, typhoid fever and pneumonia. The knowledge of the possibility of involvement of one or more joints in an acute inflammatory process in any of these diseases should keep us from error. Lack of symmetry in the distribution is a most important feature. In gonorrheal rheumatism the frequent involvement of the tendon sheaths and plantar fascia, the occasional presence of ophthalmia, with the presence of urethral discharge and the chronicity of the attack, are of value. Gonococci may be found in the exudate withdrawn from the point.

I have seen arthritis deformans come on so acutely as to cause error for a time. In children the form to which Still's name has been given is generally of slower development, and the adenitis is distinctive.

Acute arthritis of infancy, similar in appearance and probably

in causation to the infectious types described, affects a single joint usually and generally goes into suppuration. I have seen the ankle and knee successively involved with recovery after operation.

**ACUTE OSTEOMYELITIS.**—This demands extreme care in differentiation, for failure of operation is likely to mean death. The severity of the onset, and the general symptoms and local signs should attract attention. The epiphysis is involved, more commonly of the femur or tibia.

**PELIOSIS RHEUMATICA.**—The joint signs are less prominent than in ordinary acute rheumatism, being overshadowed by the purpura, exudative erythema and purpura urticans. Hemorrhage from the mucous membranes occurs.

Gout, syphilis, tuberculosis of joints, infantile scurvy and the hemorrhage into the joints in hemophilia need only to be mentioned.

Laboratory methods are of more avail in differential diagnosis from some of the diseases mentioned than in a direct diagnosis of rheumatic fever.

**Prognosis.**—This concerns: (a) Danger of life, which is not great (2 per cent.), hyperpyrexia, myocarditis, and the rare malignant endocarditis causing most of the fatalities;

(b) Liability to recurrence, which is unfavorably influenced by the youth of the patient and the family tendency;

(c) Liability to cardiac disease, which is the most important feature as to prognosis. Although a slight systolic murmur at the apex may wholly disappear and often does so, the danger of valvular disease is great in any case, and especially if murmurs have appeared, and is perhaps greater in accordance with the youth of the patient. Pericarditis is perhaps even more serious. Insurance companies commonly reject applicants giving a history of more than one attack of acute rheumatism, even though the physical examination be entirely negative.

## XII. HYDROPHOBIA

**Definition.**—Hydrophobia (lyssa; rabies) is an acute infectious disease of the dog, wolf, and other warm-blooded animals, occasionally communicated to man by bites and wounds, and caused by an

unknown specific organism. Destructive changes occur in the ganglia of the posterior roots of the spinal nerves, and in parts of the sympathetic nervous system. The poison is most abundant in the medulla.

**Etiology.**—The disease occurs alike in man and animals, and is transmissible from one to the other. Its incidence in man depends upon opportunity for its spread amongst animals, and, leaving aside the cases common in Russia from the wolf and the occasional ones in America from the coyote and the skunk, often through the intermediation of domestic animals bitten by these wild ones, the disease may be said to depend upon the presence of unmuzzled dogs in the community. In England and Germany it has been suppressed by efficient muzzling excepting when, in the former country, the ignorant and pernicious meddling of certain societies has resulted in the temporary suspension of the muzzling law, when hydrophobia has again become prevalent. In the western part of the United States the disease is propagated by the habits of domestic dogs and cats, the former occasionally associating with the infected prairie wolf and the latter with the skunk, with resulting infection. Several instances of rabies in the wild animals mentioned have been reported in this state; and many cases of infection in soldiers, cattle-men and others on the great plains have been recorded. Rats, cows, guinea pigs, and other animals may become infected.

**Symptoms.**—The period of incubation is extremely irregular and varies between two and twenty-four weeks, most commonly six to eight weeks. If the bite be inflicted by the wolf or cat, the period is likely to be shorter than in the case of a dog or other animal; if the wound be extensive and in a region such as the face, well supplied with blood and lymph vessels, the period is much shorter than if it be slight and in a less susceptible locality. The virus is apparently held in the lymphatic system for an indefinite period in certain of these cases. It is well recognized that infection does not by any means always take place after a bite by a rabid animal (15 per cent. to 20 per cent. only), and this is especially the case if the bite be inflicted through clothing or in places with comparatively rich vascular supply.

The prodromal stage is marked especially by depression of spirits, malaise, headache, slight fever, insomnia, hyperacusia, photophobia, and often by hoarseness and dysphagia. Irritation of the wound, perhaps reopening of the scar, trembling in the affected limb, shivering and loss of appetite may be noted. After a period of several days, the stage of excitement appears, with hyperesthesia of the special senses and the skin, dyspnea and characteristic dysphagia. Upon attempting to swallow a painful reflex spasm of the muscles involved in deglutition takes place. This may also be initiated by a draft of air, sudden sound, by suggestion, but especially by the attempt to drink water, or even the sight or thought of it. The thirst becomes urgent upon this account. Cyanosis may be present during the spasm. Moderate rise of temperature and pulse, irregular respiration, increased reflexes, dilated pupils, curious sounds (barking) produced in the larynx during the spasms, albuminuria, maniacal excitement, attempts of the patient to bite himself or his attendant, salivation, and occasionally death from asphyxia during a spasm, are further features of the stage of excitement. After two or three days, if the patient survive, the third or paralytic stage supervenes, with relaxation, paralysis of certain groups of muscles, very irregular as to distribution, and coma and death from exhaustion in less than a day. The absence of the second stage, more common in animals (dumb rabies) than in man, has been attributed to intensity of the infection.

**Diagnosis.**—This depends upon the history of a bite of an animal possibly rabid, perhaps with signs of irritation about the wound, and the development of the symptoms named some weeks afterward. In all suspicious cases the animal inflicting the bite should, if possible, be safely confined, the course of the disease, if any develop, should be watched, and finally examination of the cord, with animal injections, should be made, that the diagnosis may be absolutely established and proper treatment instituted.

**Differential Diagnosis.**—Lyssophobia and hysteria, in this case practically synonymous, are to be differentiated by the absence of fever, of the progressive stages of hydrophobia, and by the general emotional, neurotic and hysterical features. Landry's paralysis pre-

sents many of the features of paralytic rabies, but could cause confusion only in the possible instances in which there might be a history of a bite.

**Prognosis.**—The fully developed cases are fatal. Of persons bitten upon exposed parts, especially the face, under circumstances favorable for infection, and especially by wolves, about half may be expected to die of hydrophobia. The Pasteur treatment shows a mortality of less than  $\frac{1}{2}$  per cent. in more than 30,000 cases. All suspected cases should be sent to the institute as early as possible. Immediate cauterization of the wound probably more than doubles the chance of escaping infection.

### XIII. FEBRICULA: EPHEMERAL FEVER

**Definition.**—When no cause can be discovered for a transient febrile attack, it is designated “febricula.” A fever for a day or two only may be spoken of as ephemeral fever. Both are more common in children.

**Causes.**—Probably many transient fevers are caused by light abortive attacks of such diseases as tonsillitis, rheumatism, bronchitis, etc. Slight digestive disturbances, over-exertion, moderate exposure to heat, inhalation of putrid odors, and various unrecognized causes are doubtless operative in the same manner.

**Symptoms.**—A mild fever, but in children occasionally reaching 104° F., with constipation, headache, drowsiness, chilliness, and even convulsions in children, often beginning abruptly, with rapid fall in a day or two, or more gradual fall, if lasting longer, with scanty febrile urine, and occasionally herpes labialis, make up the symptomatology of the ordinary attack.

**Diagnosis.**—This depends upon our ability to exclude the recognized causes of fever.

**Prognosis.**—This is, of course, good.

### XIV. INFECTIOUS JAUNDICE

**Definition.**—Infectious jaundice or Weil’s disease is an acute infectious disease characterized by chill, fever, and jaundice, and

probably caused by the *proteus fluorescens* (Jäger). The disease has hardly established its position as independent of other varieties of benign jaundice, and further investigation of its supposed bacterial origin, and study of other forms of jaundice will be necessary to this end. Many cases undoubtedly belonging in other categories have been reported as Weil's disease, such as abortive attacks of typhoid, especially with gall bladder symptoms, sepsis, etc. Further, the clinical picture of the disease varies greatly in different outbreaks, and as given by different authors.

Enlargement and cloudy swelling of the spleen with jaundice, and occasionally the features of an acute nephritis, have been found post mortem.

**Etiology.**—Jäger cultivated the *proteus fluorescens* from the urine of the living and the organs of a patient dying of the disease. He believed the infection to have taken place through water swallowed while swimming in an infected stream.

The disease occurs chiefly in young men, and butchers have frequently been infected. Laborers in sewers, soldiers, and tanners have also been reported as suffering in different epidemic outbreaks. These have usually appeared in summer.

**Symptoms.**—Nothing is reported as to the period of incubation. The disease begins abruptly with a chill and high temperature, as a rule, although mild prodromal symptoms are occasionally mentioned. The fever is of the remittent type, and lasts one to three weeks in average cases, although death has occurred in two or three days. Termination by lysis is the rule. Nausea, vomiting, and diarrhea are frequently present; and headache, vertigo, stupor, delirium, coma, and convulsions may be noted. The jaundice appears in the middle of the first week, and may be deep, with clay-colored stools. The liver and spleen are enlarged and often tender upon pressure. The urine is albuminous, and a definite acute nephritis may be present.

Epistaxis, hemoptysis, petechiæ, and hematuria may occur. Muscular pains and loss of weight are notable features of the severe types. Relapse is said to occur in forty per cent. of the cases, generally within a week of defervescence, and milder than the original attack.

**Diagnosis.**—This depends upon the recognition of an attack of

acute jaundice with symptoms of acute infection, much more severe than catarrhal jaundice, but as much less severe than acute yellow atrophy of the liver. We may hope for assistance from the laboratory in the diagnosis. Unless a definite infectious agent be established, it is doubtful if the diagnosis will ever rest on a sure foundation. Wilson states that "it is probable that some of the local epidemics reported as catarrhal jaundice of severe type have been outbreaks of Weil's disease," and this seems very probable.

**Prognosis.**—Most of the cases terminate in recovery, but fulminating cases have died in forty-eight hours (Hughes).

## XV. MILK-SICKNESS

**Definition.**—Milk-sickness (trembles) is a disease occasionally caused in man by the use of the milk and flesh of cattle suffering with the "trembles." Its extensive development was a serious matter in the States west of the Alleghanies during their early settlement, but it has practically disappeared during recent years. Jordan and Harris have studied a local epidemic in cattle near Carlsbad, New Mexico, and have isolated the *Bacillus lactimorbi* as the causative organism.

The disease commonly occurs in the spring and autumn months. No immunity is established, but on the contrary one attack seems to predispose to subsequent ones.

**Symptoms.**—The incubation period is probably from two days to eight or ten days. Anorexia, headache, and fatigue are followed by nausea, severe attacks of vomiting, and even by hemoptysis, with abdominal pain as a prominent feature. Constipation is present.

Thirst, fetid breath, hiccough, dyspnea, and dysphagia are noted. Restlessness, stupor, and even coma and convulsions may occur. In severe cases the patient passes into a typhoidal state, though the fever remains very moderate (99° F.). Acceleration of the pulse is present only in the severer cases, when marked prostration is a feature. The illness lasts from a few days in the mild cases to several weeks in the severer ones, and the course may be a chronic one, according to Yandell and Kimmell.



**Diagnosis.**—If we are to accept the report of Jordan and Harris as conclusive, the diagnosis in man is likely to be established by laboratory procedures in future cases.

**Prognosis.**—The prognosis as to recovery seems to be good in nearly all cases.

## XVI. ROCKY MOUNTAIN SPOTTED FEVER

**Definition.**—Rocky Mountain spotted fever (tick fever) is an acute infectious disease, transmitted by the tick, *Dermacentor occidentalis*, but so far without established bacterial cause, although Anderson believes the *Piroplasma hominis* to be the agent of infection. The disease is transmitted to laboratory animals through inoculation.

Tick fever occurs chiefly in the valley of the Bitter Root River in western Montana, but a few cases have been reported from Idaho, and scattering ones from Wyoming, eastern Oregon, Nevada, and Utah. Four or five cases have been recognized in Colorado. The cases have practically all occurred at elevations greater than 3,000 feet, and during the spring and early summer, in April, May, and June especially. Age and sex seem to cut no especial figure, excepting as to the chance of exposure.

The disease occurs in those exposed to the bite of the tick, hence especially in ranchmen, lumbermen, surveyors, etc. Seventy-six of Anderson's one hundred and twenty-one cases were in males.

**Symptoms.**—The period of incubation is thought to be about one week. Chill or a series of chills, sharp fever, nausea, vomiting and the usual manifestations of an acute infection appear. The temperature reaches 104° or 105° F., with morning remissions, and in severe cases continues high for two or three weeks. In milder cases distinct intermissions may occur, as many as six or seven paroxysms of fever of two or three days duration, separated by intermissions, being noted. The first attack in such cases is likely to be the most severe.

The eruption appears generally in three, four, or five days, at first about the wrists, ankles, and forehead, and spreading thence over the body, but less abundantly. The rash is maculopapular and

then hemorrhagic in typical cases, and in severe cases ecchymoses appear. The rash may be absent in mild cases. It tends to disappear with the defervescence, and scaling may follow. In a Wyoming case now recovering in St. Joseph's hospital, the eruption over the trunk is nearly as abundant as in measles.

The spleen is enlarged and often tender, and the liver less frequently so. Jaundice may be present. The pulse is rapid and feeble, and the pulse rate is high in severe cases, especially if bronchitis be present. Albuminuria and even acute nephritis may be noted. Many red cells are destroyed, resulting in a mild secondary anemia. A moderate leukocytosis is reported, and the large mononuclear cells are said to be increased. Gangrene of the fingers, toes, and male genitals has been noted.

**Course.**—In mild cases defervescence occurs by the eighth or tenth day, and in severe ones during the third week. In the worst infections no improvement is noted and bronchopneumonia, nephritis, cardiac exhaustion, or other complication appears as a forerunner of the fatal result.

**Diagnosis.**—This depends chiefly upon the history of exposure to the tick, frequently upon finding the tick upon the body or limbs, in those regions where the infection is known to exist. In case of doubt, examination of the blood for septic organisms would be of assistance in excluding pyemia and septicemia, and of the spinal fluid in excluding cerebrospinal meningitis.

**Prognosis.**—The fatality varies greatly with the locality and the season. In certain outbreaks, three-fourths of those affected have died, but the average mortality in Montana is stated to be twenty per cent. or less. It is lower in the other States mentioned.

## XVII. FOOT-AND-MOUTH DISEASE

**Definition.**—Foot-and-mouth disease (aphthous fever) is an acute infectious disease of cattle and sheep, occasionally attacking other animals. While readily transmissible to man, it usually occurs in the human race only sporadically.

**Etiology.**—The identity of the infectious agent has not been

established unless it should develop that the micrococcus isolated from the lesions of the disease by Klein should prove to be the specific organism.

The prominent feature in the etiology is the occurrence of epizootics, especially in cattle, and the opportunity for infection of man through contact with the cattle, their quarters, food, etc., and their excretions. It is well established that the infection may be carried in milk and its products, but infection through the meat of infected animals has not been proven. The use of unsterilized milk is without doubt the common means of transmission. In the recent extensive epizootic in Pennsylvania and New York, no case of infection in the human family was reported.

**Symptoms.**—The incubation period is from three to five days and slightly longer in some cases. Chilly feelings and moderate malaise mark the onset, with soreness about the lips and mouth, similar to that of a mild stomatitis. Reddening of the mucous membrane, fetid breath, difficulty in swallowing, and occasionally vomiting, vertigo, and insomnia are present. The vesicles appear on the tongue and mucous membrane of the mouth and lips, and may form pustules, and even extend to the face. Small ulcers are left after the pustules rupture, healing with a scar, but the vesicles undergo dessication and heal directly. Roseolous and urticarial eruptions may appear. Nose bleed is not uncommon, and other hemorrhages may be noted. Recovery usually takes place in one or two weeks.

**Diagnosis.**—This depends upon a knowledge of the opportunity for infection in association with the features mentioned. The ready transmission of the disease to the lower animals (calf) may be taken advantage of in case of doubt. The various forms of stomatitis must be considered, but none of them conforms to the clinical course outlined. The occasional infection of the fingers may be valuable in diagnosis.

**Prognosis.**—In adults it is good. In children, owing to the difficulty of taking food, and the possibility of septic infection in the ulcerations, the outlook is more serious, and death occasionally results.

## XVIII. MILIARY FEVER

**Definition.**—Miliary fever (sweating sickness) is an acute infectious disease of unknown origin, and characterized clinically by fever, sweating, and a profuse miliary eruption. It was very common in the fifteenth and sixteenth centuries, and apparently more fatal than in recent years. The disease has appeared in epidemic form in the past twenty years in England, and several Continental countries, but not, so far, in the United States.

**Etiology.**—Although it is recognized that young women and children more often contract the disease, and that it occurs in limited epidemics, nothing definite is known of its etiology.

**Symptoms.**—The incubation period has not been established. After a short prodromal period, fever and sweating appear, with epigastric distress. On the third or fourth day an erythematous rash appears, followed by a copious outbreak of miliary vesicles, most abundantly upon the neck and trunk. In forty-eight hours desquamation may begin. In the severe forms delirium and hemorrhage may be noted, and all the signs of an intense infection, ending fatally. The disease lasts a week or two in the milder cases, but three or four weeks in the severer ones. Relapse may occur.

**Diagnosis.**—Owing to the extent of the epidemics in certain instances, influenza and dengue would be strongly suggested, for each of these diseases affects a large percentage of the population. Influenza is a common and familiar disease, and without the extensive miliary eruption, while dengue has a totally different geographical distribution. I have seen the body absolutely covered with miliary vesicles in acute rheumatism and in pneumonia, but confusion could not occur.

**Prognosis.**—This was grave in many of the epidemics described in history, and the mortality has been as high in the course of some recent epidemics, death even occurring on the first day. In general the prognosis has been favorable in the epidemics of the past twenty or thirty years. With the improvement in hygienic conditions everywhere it seems probable that this disease is losing its virulence.

### XIX. GLANDULAR FEVER

**Definition.**—This is an acute infectious disease of children, of unknown origin, and recognized clinically by the notable enlargement of the cervical lymph nodes.

**Etiology.**—Aside from the fact that glandular fever occurs in small endemics or epidemics in children, and in cold weather, nothing is known.

**Symptoms.**—The incubation period is thought to be from five to eight days. A sharp but moderate rise of temperature occurs ( $103^{\circ}$  F.) with the usual phenomena of infection in mild degree. Vomiting and delirium may occur. A mild angina is followed by swelling and tenderness of the cervical glands, which may reach the size of an olive, with surrounding edema in severe cases. The axillary and inguinal nodes may be involved. If bronchitis develop, enlargement of the tracheobronchial glands occurs, and severe dyspnea may result. Suppuration of the glands is extremely rare. Splenic enlargement has been noted.

The fever lasts about two weeks, the glands gradually resolving after defervescence. Otitis media and similar mild complications may occur, and nephritis and retropharyngeal abscess have been reported.

**Diagnosis.**—This rests upon the exclusion of other causes of adenitis.

**Prognosis.**—All cases recover unless some unusual complication develops.

### XX. SWINE FEVER

A painful type of migrating erythema, with adenitis, swelling of the affected fingers and the development of small nodules is occasionally seen in those who dissect sick pigs, but the exact bacteriology is unknown.

### XXI. RAT-BITE FEVER

Most of our knowledge of this rare condition comes from Japan. After the injury the period of incubation may extend over several

months. The wound, often apparently healed, becomes inflamed and ulcerated, and the lymph glands are involved. A chill, high fever, and erythematous eruption are noted, with muscular pains, delirium, etc. After a few days the symptoms subside, to recur after weeks or months, perhaps several times. Recovery is the rule. The infectious agent is still in dispute.

## SECTION II

### DISEASES OF THE BLOOD

**Anemia.**—By this term is meant a condition of the blood in which there is a reduction in either the red cells or the hemoglobin or both, or the total quantity of blood. With the leukocytes we are not especially concerned in the diagnosis of anemia. Local anemia may be present in a certain part of the body, as under the influence of cold, disease or perverted vasomotor function. We are especially concerned with the general anemia in which the blood of the whole body is affected.

#### 1. PRIMARY ANEMIA

In this type some defect in hemogenesis exists so that a proper amount of normal blood is not formed.

##### A. CHLOROSIS

This is an anemia of unknown origin occurring in the female sex, rarely in males, early in life and characterized by a moderate diminution in the number of red cells and a greater decrease of the percentage of hemoglobin.

**Etiology.**—The defect in the blood-forming apparatus is not known. The disease is found in girls at and soon after the time of puberty, and may extend to the third decade. It is especially subject to relapse. It is particularly common in newly arrived immigrants, and in girls moving from the country to the city, but seems from Cabot's conclusive investigation to be less common than formerly, less common even than pernicious anemia. Girls in domestic service suffer much more frequently than those in shops and factories. It is comparatively rare in the well-to-do. Poor air, poor

food, bad habits as to eating, hard work, and especially chronic constipation are of importance in the etiology. The relationship between menstruation and its disorders and chlorosis is not clearly established. The hypoplasia of the circulatory and genital systems mentioned by Virchow is found only in a small minority of the cases. The disease seems to be hereditary in certain families, and is more common in blondes than in brunettes.

**Pathology.**—No definite statement can be made upon this point. The rarity of opportunity for post-mortem examination in chlorosis may in part account for this.

**Symptoms.**—The disease comes on so gradually that no date can be set for its beginning in most cases. The patient complains of weakness, of dyspnea and palpitation upon exertion, and various dyspeptic symptoms, pain in the epigastrium and cardiac regions being not uncommon. Attacks of fainting are frequent, the ankles are often puffy and the skin about the eyes is occasionally affected, giving a full plump look, strangely in contrast with the sickly yellowish-green color of the typical cases. When this color is typical it is pathognomonic. One may be deceived by the lack of pallor, but in such cases the blue sclerotic, and the delicate type of complexion, with the symptoms noted, should compel an examination of the blood. The patients frequently crave strange things to eat; and chalk, dirt and slate pencils are perhaps taken as often as the more commonly mentioned pickles.

**Physical Examination.**—The color of the skin has been mentioned; occasionally pigmentation over the joints is noted. The subcutaneous fat is generally well preserved. Slight enlargement of the heart, and hemic murmus, systolic in time and heard at almost any portion of the precordia, are commonly present. The loud venous hum in the neck is found nearly constantly if carefully sought for. The disappearance of the cardiac murmurs upon the administration of iron is sufficient evidence of their non-organic character. The examination of the lungs is commonly negative, but care should be taken to exclude pulmonary tuberculosis. The spleen is occasionally enlarged. Dilatation of the stomach and gastroptosis are not infrequent, and the low right kidney is common. Hyperchlorhydria



is occasionally present. A trace of albumin and a few hyaline and granular casts are often reported. Transient febrile movement may be found and does not at all point to pulmonary involvement.

**The Blood.**—The typical chlorotic blood shows approximately four million red cells, with considerable latitude for variation, and a hemoglobin content of less than 50 per cent. in most of the cases. Cabot found the color index below 5/10 in 3/5 of his series. The white count shows nothing characteristic. The pallor of the blood drop is evident to the eye. The stained slide shows marked variations from the normal in size and color of the red cells, the average cell being smaller and definitely paler from its lessened content of hemoglobin. Marked poikilocytosis is not often seen. Nucleated red cells, of the normoblastic type are occasionally found. The whites, as in the blood count, show nothing characteristic in the blood slide. Haldane and Smith report an increase in the total volume of blood—a polyplasmia, but no definite meaning is thus far attached to the finding.

Hysterical features are not uncommon. A serious complication is the occasional venous thrombosis, generally seen in the legs, but occasionally in the cerebral sinuses. The danger of pulmonary embolism, which may be fatal, should be noted.

**Diagnosis.**—This depends upon the recognition of the anemia in a girl or young woman, with the characteristics as shown by the blood examination mentioned above. Hysteria and neurasthenia must be considered, and pulmonary tuberculosis excluded. The possibility of an anemia, secondary to unrecognized loss of blood, as from hemorrhoids or from intestinal parasites or malaria, must not be overlooked. Many of the patients are sent to the physician's office on account of supposed heart disease, and some with supposed nephritis, but no difficulty should arise if the blood and urine be examined.

**Prognosis.**—This is good under efficient treatment, but the tendency to relapse should be recognized and mentioned to the patient or family. The danger of cerebral thrombosis or pulmonary embolism is to be taken into consideration.

**B. PERNICIOUS ANEMIA***(Idiopathic Anemia)*

An intense anemia, usually fatal, of unknown origin, characterized by faulty production of red cells and their increased destruction.

**Etiology.**—The cause is unknown, but many supposed contributory factors may be considered. Heredity plays but a trivial part. In Cabot's 1,200 cases nearly twice as many males as females were affected, though females predominated slightly in the first half of life. More than half of all the cases occur between 40 and 60 years of age. In the first decade the disease is excessively rare, and decidedly so in the second. A few cases originate in pregnancy, and the puerperal state. It is probably less common in residents of the country. Occupation seems to have no especial bearing. Chronic diarrhea and pyorrhea alveolaris are occasionally noted in the history, and long-continued loss of blood has been thought to be of importance. Flint found atrophy of the mucous lining of the stomach, but there seems to be no good reason for thinking it to be closely related as a cause. Of cancer of the stomach we may say that it produces typically a secondary anemia, yet in some cases of supposed pernicious anemia this lesion has been found post mortem. The anemia caused by certain parasites, especially by the fish tapeworm and the hookworm, resembles the pernicious form in certain ways, but may ordinarily be differentiated. There is no very definite evidence that either nervous shock or syphilis is of especial import in the etiology. There is little doubt that the disease is vastly more common in certain sections than death returns would indicate, because of failure or lack of opportunity on the physician's part to investigate properly. Many of the patients do not consult a physician until the disease is well advanced.

**Pathology.**—The body fat is fairly preserved, but the peculiar pallor is notable even after death in certain cases. Fatty degeneration is present in the heart, kidneys and liver, the latter organ being frequently enlarged, and very yellow in color. The spleen is usually shrunken, red and sclerotic, but is occasionally much en-

larged. The increase in the iron pigment in the liver, kidneys and spleen is notable. The total quantity of blood is markedly diminished at death. The marrow of the long bones is soft and red, and megaloblasts are present in unusual numbers. A patchy degeneration affects especially the posterior columns of the cervical cord.

**Symptoms.**—The patient has commonly noted for some months an increasing debility, with palpitation, dyspnea on exertion, sleeplessness, langour, dyspeptic symptoms, slight puffiness of the ankles and marked failure of muscular strength. His friends have noticed his pallor, and the physician is forcibly impressed with the yellow tinge, which even suggests jaundice in certain cases. The ghastly color of the lips is extremely suggestive in others. Headache, vertigo, and tinnitus are frequently noted, and dimness of vision more rarely. A fair preservation of muscular strength is occasionally present.

A slight or moderate elevation of temperature is found in most cases, at least during certain stages of the disease.

Nervous symptoms are very common, and generally dependent on the degenerative lesions in the cervical cord. Paresthesias are very frequently noted, and severe pains in the course of the peripheral nerves and in the epigastric region may be present. Attacks similar to the gastric crises of locomotor ataxia are occasionally seen. The knee-jerks may be increased, suggesting that the lateral columns are especially involved, or the loss of reflexes may suggest rather a tabetic type of distribution. If the peripheral nerves are especially painful and tender the possibility of arsenical neuritis, which I have seen several times in such cases, must not be overlooked. Loss of control of the sphincters is not uncommon in the spastic type of cord involvement and may be found in certain cases suggesting a transverse involvement of the cord.

**MENTAL SYMPTOMS.**—Mental dulness, delirium, delusions, hallucinations, melancholia and dementia are noted in a few cases.

**CIRCULATORY SYSTEM.**—The heart is commonly moderately dilated, occasionally much dilated. In practically all cases systolic murmurs of hemic origin may be heard. A relative leakage at the mitral orifice accounts for many of the apical systolic murmurs.

When we consider the poor state of nutrition of the heart muscle and the poverty of the blood, with the diminution in its total quantity, we need not be surprised at the constancy of the auscultatory signs. The blood pressure is greatly reduced, perhaps to 60 or 80 mm. The vessels of the neck may pulsate so violently as to suggest aneurism, and the pulse is often of a jerky character. Petechial hemorrhages are present in many cases, and retinal hemorrhages in a majority.

**The Blood.**—It is paler than normal and flows quickly into the paper used in some of the tests for the hemoglobin. Because of the relative increase in the hemoglobin, to be mentioned later, the color of the blood is relatively better, as compared with the red count, than in cases of chlorosis. The red cells are strikingly reduced in number, and most of the cases have a count below two millions, while a considerable portion fall below one million. Cabot's point is well taken that there is no other common disease of temperate climates that reduces the red count to two millions or below. The hemoglobin is typically present in more nearly normal proportion than the number of red cells would indicate, so that the color index is commonly 1.0 or somewhat greater. As the patient improves, the number of cells increases more rapidly than the hemoglobin percentage, so that the color index falls, often to slightly less than 1.0. The abnormal number of large cells containing an excess of hemoglobin accounts for the high color index.

In the stained slide the most striking feature is the marked poikilocytosis, beyond that seen in any other disease. The cells vary much in size and many are below the average. Nevertheless the most characteristic feature according to Cabot, is the presence of oversized cells. Anomalous coloring of the red cells is practically constant and a well-defined polychromatophilia is noted in most cases. Nucleated red cells are a constant feature of pernicious anemia, the relative proportion varying much in the different stages of the disease. Both megaloblasts and normoblasts may be found in almost every case if persistent search be made, and they may increase enormously in number in the so-called blood crises. The leukocytes and the blood platelets are commonly reduced in number,

the latter markedly so. A small percentage of myelocytes is not uncommonly present. The urine is often of low specific gravity, but normal in quantity, and presents no characteristic diagnostic features. A trace of albumin or an increase in uric acid may be noted. In one of my cases the urine was increased coincidently with an unexplained increase in the blood pressure.

**Course.**—The disease is generally chronic, extending over several years, but acute cases, fatal even in a few weeks, are not unknown. The frequency of remissions, in which the patient appears to have absolutely recovered, should be noted. One of my patients under the usual arsenical treatment showed during a remission five and one-half million red cells and a practically normal blood picture, suffered an acute suppurative appendicitis at this time, made a normal recovery after operation, but died in relapse within a year. One physician in the fourth year of the disease returned to an eastern city and resumed his practice, but died within a year after. The probability of a relapse in any given case, no matter how favorable, must never be forgotten, and from two to even five or six recurrences are not infrequent.

**Diagnosis.**—This is generally easily made upon a consideration of the peculiar color, symptoms indicating a marked anemia, frequent history of relapse, absence of history of severe hemorrhage, or other reason for the blood condition, and the finding of a low red count with a relatively high hemoglobin percentage, with poikilocytosis, and other features mentioned above. The disease must be differentiated from the secondary anemias, especially those seen in connection with persistent losses of blood, the presence of intestinal parasites or cancer of the stomach. These conditions are generally capable of diagnosis, if care be used, even without study of the stained slide. The low color index of secondary anemia, the greater average number and smaller average size of the red cells, their central pallor when stained, and predominance of small nucleated reds over the larger forms, are decisive. The cord lesions may produce symptoms which lead the patient to see the neurologist rather than the internist, but the lack of a complete picture of the systemic disease of the cord commonly imitated and the study of the blood, suffice to prevent error.

**Aplastic Anemia.**—In this subvariety certain features demand attention. It affects young females especially, and is acute, progressive and quickly fatal. The marrow is found, post mortem, to be fatty and aplastic. Hemorrhages are much more frequently seen than in the typical pernicious anemia, and purpura may be present. The color index is low, the lymphocyte percentage is relatively high, and nucleated reds are absent. The essential feature is a loss of the blood elements having their origin in the bone marrow.

**Prognosis of Pernicious Anemia.**—Probably less than 1 per cent. recover, and not for a considerable number of years is relapse to be considered improbable, regardless of the good condition present.

Most of the cases die within three years, many within one, while a fair number live for a varying period, with remissions of considerable length, a duration of even 10 or 15 years being occasionally recorded.

## 2. SECONDARY ANEMIA

### *(Symptomatic Anemia)*

Secondary anemias are those which follow upon some well-defined cause. They may be acute, as after violent hemorrhage, or destruction of a large proportion of the red cells by virulent malaria, or after septic affections, or as the result of hemolytic poisons, of which nitrobenzol may be taken as typical. The chronic forms are found in many chronic diseases in which blood is lost persistently, or there is an inability of the blood-forming organs to make blood. Thus persistent small hemorrhages, as especially from internal hemorrhoids and uterine fibroids, may induce marked secondary anemia. Interference with the taking and digestion of food, as in cancer of the esophagus or other portions of the digestive tract, is a common cause, often combined with loss of blood from ulceration. Pure starvation may be the sole cause. The intestinal parasites, notably the hookworm, and the *Dibothriocephalus latus*, induce anemia, the latter variety approaching the pernicious type. Loss of blood from intestinal lesions and a chronic toxemia are probably both operative in the hookworm infection, while the latter is the main factor in

the fish tapeworm infection. Anemia also follows acute infectious diseases and is found in chronic diseases, such as syphilis and malaria, chronic nephritis, cirrhosis of the liver, etc. Lead and other mineral poisons are frequent causes. Prolonged suppuration, of the type that induces amyloid degeneration, and prolonged lactation should be mentioned. The anemia associated with sarcoma of the kidney in babies, and with rapidly growing cancer in adults, may be very marked.

**Symptoms.**—These do not differ from those found in the essential anemias already described. The very rapid induction of anemia by hemorrhage, or by an overwhelming malarial infection, produces more acute symptoms than are described heretofore, but the main characteristics do not otherwise differ. The fainting and convulsions from sudden loss of blood, as, for example, in ulcer of the stomach, are to be noted. Loss of one-half of the blood of the body suddenly is an extremely dangerous if not fatal accident. Headache and other symptoms induced by poor supply of the blood to the brain, faintness, dyspnea, palpitation, amenorrhea, dyspepsia, etc., are common in all anemic conditions. The lack of energy, long attributed to laziness in the inhabitants of the hookworm infected region, is typical of the langour and debility resulting from anemia.

**The Blood.**—As soon as the fluid portion of the blood is recouped after a sudden hemorrhage, a marked reduction in the hemoglobin and the number of red cells may be noted. Because of the lack of the hemoglobin in the regenerated cells the color index is low. In an ordinary secondary anemia two, three or four million cells are found with perhaps only half or two-thirds of the appropriate percentage of hemoglobin, so that a color index of about 5/10 to 7/10 is very often found. Poikilocytosis of moderate degree is present, and the reds are notably pale. A moderate number of normoblasts is found, and a temporary increase in the polynuclear white cells is often present.

**Diagnosis.**—This depends upon the recognition of the symptoms enumerated, in conjunction with the blood findings given. It is well to assume that every anemia is secondary until careful consideration suffices to exclude any of the known causes.

**Prognosis.**—This obviously depends upon the cause. Transfusion saves many acute cases, a checking of loss of blood or of some chronic cause of intoxication may save many more, while those depending upon chronic advancing disease, as cancer, nephritis, etc., are hopeless.

### 3. ERYTHREMIA

#### *(Erythrocytosis Megalosplenica)*

This is a rare disease in which great increase in the number of erythrocytes is present, generally in connection with cyanosis and enlargement of the spleen. The condition of primary tuberculosis of the spleen noted in early cases is not a frequent one. Lucas gives the best summary of the reported cases.

**Etiology.**—It is a disease of middle age, and about equally distributed between the sexes, but the cause is unknown.

**Pathology.**—Osler believes that the disease is due to “a primary hyperplasia of the erythroblastic bone marrow.” The viscosity of the blood is increased, this being of interest in connection with the enlargement of the smaller vessels. The marrow may be intensely purple. The spleen is firm and moderately enlarged in most cases.

**Symptoms.**—The patient complains of headache, dizziness, and weakness, and occasionally of bleeding at the gum margin. Upon examination a degree of cyanosis, of the face and lips especially, may be noted, passing beyond almost any other such condition compatible with ability to be out of bed. The lips and tongue, as in my case, may be of the exact shade of a ripe concord grape. The cyanosis is darker in winter, more dull reddish in warm weather. Osler speaks of a vasomotor instability, such that the hands become engorged if held down, but anemic when elevated. A painless, hard enlargement of the spleen of moderate degree is present in most cases, but was absent in my own. Edema of the lung may be noted. Albumin and casts in the urine are found in many cases. Jackson reported the retinal veins enormously dilated in the case referred to. The blood is increased in total volume. The most characteristic



feature of the disease is the increase of the red cells to 7 to 12 millions per cubic millimeter, with a hemoglobin content far above the normal (even 200 per cent.). Moderate increase in the leukocytes is reported in most cases, with relative increase in the polynuclear cells. Normoblasts and myelocytes may be found. The blood pressure is generally raised. Amongst other symptoms may be mentioned muscular weakness, flushing, hemorrhage into the skin, constipation and fainting attacks. Ascites has been found, probably connected with the splenic enlargement.

**Diagnosis.**—This depends upon the finding of the symptoms mentioned and the exclusion of other causes of cyanosis and polycythemia. The latter condition in high altitudes is not accompanied with blueness, even though 6 or 7 million reds be present, but the cyanosis of congenital heart disease may have a decidedly increased red count. The cyanosis from acetanilid and other coal-tar products should always be thought of before giving any consideration to other forms, for it is very common in recent years and often shows a polycythemia.

**Prognosis.**—Recovery is not to be expected, but the patients sometimes live for years in reasonable comfort. Cardiac exhaustion is the most frequent cause of death.

#### 4. LEUKEMIA

**Definition.**—A disease characterized by great increase in certain of the leukocytes of the blood, with hyperplasia of the tissues which form them. It may be acute or chronic.

**Varieties.**—Most cases may be definitely classed as (a) lymphoid or lymphatic leukemia, the lymphocytes being increased in number and the lymphoid tissues being chiefly involved; (b) splenomyelogenous leukemia, the excess of white cells being largely due to myelocytes, and the pathological changes in the blood-forming tissues being more prominent in the spleen and the marrow. Recent reports indicate that spleen, marrow and lymphatic glands are involved in every case, though to a varying extent. In lymphatic leukemia the marrow may be so transformed as to resemble closely the tissue of the lymphatic glands. A myeloid transformation of lymphatic glands

is likewise seen in splenomyelogenous leukemia. Acute and chronic types are also distinguished. Cases not capable of classification as above may be termed atypical leukemias.

### LYMPHOID LEUKEMIA

**Etiology.**—The cause is unknown. It is distinctly less common than the splenomyelogenous form. The acute type is more frequent in the early decades of life, the chronic in the later. Males are more often affected than females. It has been noted occasionally in X-ray operators.

**Symptoms.**—**ACUTE TYPE.**—The most severe form develops much after the mode of a malignant tonsillitis, with ulcerations about the gums and tonsils, hemorrhages in these regions, from the nose, or under the skin, swelling of the glands of the neck, rapidly progressing anemia, utter prostration, high fever, and a malignant course and fatal termination. I have seen two such cases fatal during the second week.

**CHRONIC TYPE.**—A milder course without throat involvement, but with weakness, anemia, prostration and a more chronic type of adenitis is much more common. The chronic form is much milder and is seen in older patients. General enlargement of lymph glands, slight enlargement of the spleen, and anemia with weakness are more prominent features. Pigmentation of the skin and development of lymphoid tumors in the skin are mentioned. Mild digestive difficulties are not unusual, and hemorrhages may occur from almost any of the usual sources, even retinal hemorrhage.

**Physical Examination.**—Moderate or great increase in the size of the glands of the neck, axillæ and groins is generally present, and the abdominal and bronchial glands may be enlarged. The liver is slightly or moderately increased in size in a majority of cases. In one of my cases the liver had been aspirated unsuccessfully by two different surgeons under the belief that an abscess must be present, and peritoneal friction over the liver was a source of much distress. Such marked hepatic involvement is unusual.

Loss of weight, moderate fever, a trace of albumin and a few

tube casts, general pruritis, deafness and exophthalmos are other features which may appear.

The viscosity of the blood is commented upon by most hematologists, due to the presence of the great number of white cells. The red count may be expected to fall within the limits of one and four millions in most of the cases, the average of Cabot's cases when first seen being 2,800,000. The color index is generally low early in the disease, but above normal after pronounced anemia occurs. The white count is very high in most cases, but the average is far below that in the splenomyelogenous variety (180,000 as against 410,000 per cubic millimeter respectively, Cabot). Although a very high count is possible it should be made very plain that an enumeration of only 10,000 to 30,000 may be present in a perfectly clear case, rendering a differential count of the leukocytes necessary to the diagnosis. The case with the large liver mentioned above had a count below 15,000 when first seen. The striking feature is the predominance of the lymphocytes, over 90 per cent. in many cases. The small cells are more often found than the large, are more frequently predominant in the chronic cases, but small ones may be abundant in the acute form. Large lymphocytes were abundant in both acute cases quoted.

#### MYELOID LEUKEMIA

The disease is not very common, though much more frequently met with than the lymphoid form. We have no definite knowledge of its causation. Males are more prone to the disease, and it occurs preferably in the mid-term of life. Several cases have been reported in the same family.

**Pathology.**—The changes as found post mortem are much more striking than those, chiefly in the hemopoietic system, found in the lymphoid form. The blood is increased in quantity and many of the internal veins are widely distended. The pus-like appearance of the clotted blood has long attracted attention. The spleen presents a chronic hyperplasia, with great thickening of the capsule, and enormous increase in weight, often five to ten or fifteen pounds. The fat in the marrow of the bones is replaced by a grayish tissue con-

taining myelocytes and nucleated red cells. The liver may be much enlarged through a diffuse infiltration or the overfilling of its abundant capillaries with leukocytes, or definite leukemic tumors may be present. These may occur in other internal organs and in the skin. A moderate enlargement of the lymph glands is present in most cases.



FIG. 36.—SPLENIC LEUKEMIA. Edema of genitals from interference with venous return.

**Symptoms.**—Abdominal enlargement, loss of weight and strength, dyspnea, palpitation, hemorrhage from the nose or elsewhere, or diarrhea, are the common symptoms which lead the patient to seek advice. Inability to fasten the corset was the cause in a school teacher whom I saw. A sudden hemorrhage may be the first serious symptom. Marked anemia may be present at the first examination, with a history generally of gradual failure of health. Chills are less common than a moderate, variable fever. The heart may be dilated, more commonly displaced upward by the enormous spleen. The pulse in-

creases in frequency. As the disease progresses a majority of the patients have hemorrhages from the nose, stomach, kidneys, lungs, gums, or under the skin. As the heart weakens, dropsy of the legs or general anasarca may appear. The hemorrhagic tendency may lead to extensive hemorrhagic retinitis. Dyspnea from mechanical difficulties in breathing is rendered worse by the weak heart action, deficiency of red cells and hemoglobin, and by the filling of the capillaries of the lung with the leukemic cells. Cough is not frequently troublesome. Priapism and edema of the genital organs are occasional features. Deafness may develop. No characteristic changes occur in the urine excepting the increase in the uric acid. A few patients complain bitterly of pain from perisplenitis or perihepatitis.

The enormous spleen is the most prominent feature found upon physical examination. The notches are distinctive. The patients with diarrhea show a diminution in the splenic tumor during the attack. The enlargement of the liver may practically always be demonstrated. That of the lymphatic glands is generally a very moderate one. Leukemic infiltration of the skin, lips, mammae, etc., is occasionally noted.

**The Blood.**—Examination of the blood is necessary to the diagnosis, and in typical cases the diagnosis may be based upon the blood examination alone. The white cells may number anywhere from 50,000 or 60,000 to well over a million, 410,000 being the average quoted by Cabot. All types of cells are increased absolutely, and there are, in addition, from 20 per cent. to 50 per cent. of myelocytes, the abnormal cells which characterize the disease. Most cells and eosinophiles are much increased in number, many eosinophilic myelocytes being present. The red cells do not show marked decrease until the disease is well advanced, dropping to three or four millions and later to even one or two millions. The color index is low, as in the secondary anemias. The presence of nucleated red cells, large and small, is characteristic of leukemic blood, even though marked anemia be not present. The blood plates are increased in number.

The great number of very large white cells is a striking feature

of the blood picture, and the first impression is apt to be that the white cells outnumber the red ones. As a matter of fact they are generally present only in a proportion of 1 to 5 or 1 to 10. In a case seen at St. Joseph's Hospital the whites, just before death, actually outnumbered the reds.

**Diagnosis.**—It is practically unmistakable in the splenic form if the blood be examined, even without staining. In the lymphoid variety with many small lymphocytes the diagnosis is equally easy, while if the white count falls to 10,000 or 20,000, error is easily possible without a very careful differentiation of the white cells. In one of Cabot's cases a pneumonia following whooping-cough presented a small lymphocytosis of 75 per cent. in a full white count of 94,000. Error was probably unavoidable in such a case. In hereditary syphilis with active manifestations I once made a similar error, but the specific treatment restored the child to complete health. Sepsis is stated to produce a lymphocytosis instead of a polynucleosis in rare cases.

**Prognosis.**—Recovery probably never occurs. The child of seven years with the large liver, quoted above, grew to manhood, still having a moderate excess of small lymphocytes. His improvement under arsenic has been remarkable, but I have seen no other result comparable to this. An acute case with increase in the large lymphocytes and hemorrhage from the gums and tonsils, in which I gave a fatal prognosis, recovered sufficiently under arsenic to return to his work and was alive a year later, and in fair health, but with marked lymphemia. A splenic case died from hemorrhage from aspirating the spleen, the physician thinking an abscess existed. In general the acute lymphoid cases die within a few months, the chronic ones living several years. The splenic cases in general are intermediate in severity.

### ATYPICAL LEUKEMIA

Under the title "Atypical Leukemia" Cabot mentions:

"1. Leukæmic changes in the hæmopoietic system without leukæmic blood (pseudoleukæmia).

"2. Leukæmic blood without leukæmic changes in the blood-making organs (circulating myelomatosis).

"3. Apparent combinations of leukæmia with pernicious anemia (leukanæmia).

"4. Tumor-like growths of hæmopoietic tissue with or without leukæmic blood: (a) Diffuse (chloroma, Sternberg's leukosarcoma); (b) local (myeloma)."

**Leukanemia.**—Leukanemia is a rare condition in which a combination of the symptoms and the pathological findings of leukemia and pernicious anemia is present in the same individual. Cabot believes that they are cases of "leukemia with terminal anemia or pernicious anemia with lymphoid or myeloid marrow."

**Chloroma.**—Chloroma is an atypical form of leukemia with lymphatic tumors, found especially in the bones of the face and skull, of greenish color upon section. Marked deformity is produced, and exophthalmos, deafness, blindness, and severe pain often develop. Infiltrating lymphoid tumors may be found elsewhere. The blood shows the features of acute lymphoid leukemia. The disease is generally fatal within a few months. Sarcomatous and malignant adrenal disease with metastases to the orbit and cranium must be differentiated.

## 5. HODGKIN'S DISEASE

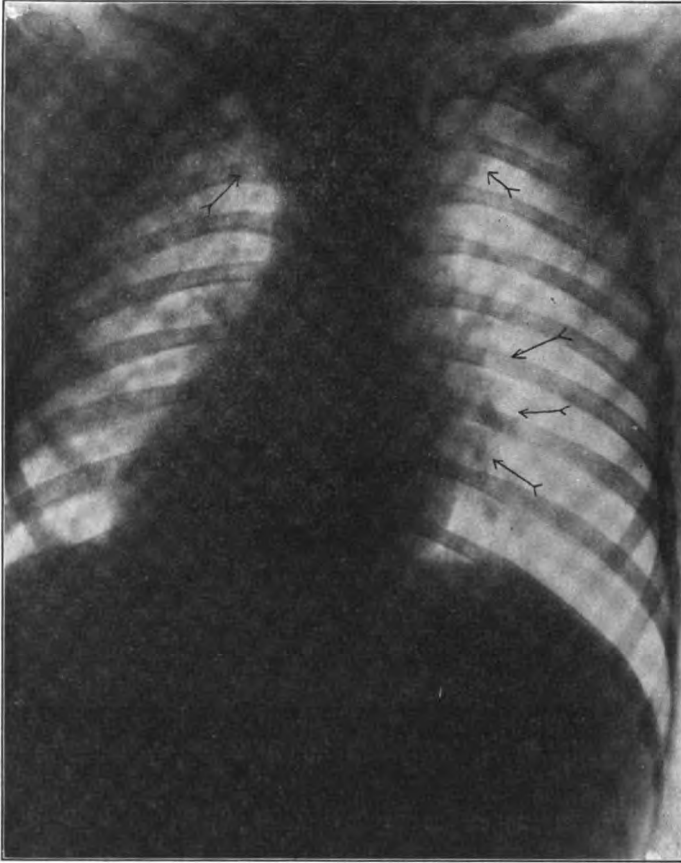
(*Pseudoleukemia*)

**Definition.**—A progressive type of anemia characterized by marked enlargement of the lymph glands and occasionally of the spleen.

**Etiology.**—The cause is unknown. The disease occurs more frequently in adult males than in other individuals. Gibbon inclines to the belief that it is malignant in nature, to be classed with malignant tumors. No satisfactory conclusions are to be drawn from the extensive bacteriological studies of recent years. Spirochetes have been found in the glands in certain cases. The theory that the disease is definitely tuberculous has lost ground.

**Pathology.**—The lymph glands are found to be enormously en-

larged, both the external and internal groups. They gradually become firmer and harder, but generally remain discrete. The capsule may give way and the adjacent tissues become infiltrated. Entire absence of caseation is notable. The spleen is generally moderately enlarged, and the lymphoid tissues of the entire body eventually be-



· FIG. 37.—HODGKIN'S DISEASE OF THE MEDIASTINAL GLANDS. (Dr. G. H. Stover.)

come affected. Nodules appear in the liver, kidney, lungs, marrow, tonsillar tissues, and skin. In one of my cases the breast had been amputated because the pseudoleukemic infiltration had been regarded as a malignant growth.

**Symptoms.**—The first complaint is generally of enlargement of



the superficial lymph glands, which become uncomfortable or unsightly. Malaise, fever, or tonsillitis may bring the patient to the physician's office. The cervical glands are commonly first enlarged, frequently upon one side only, and the axillary, inguinal, bronchial, mediastinal, and retroperitoneal glands follow in order in average cases, according to Gowers. In the neck the glands of the posterior triangle are generally first involved, the glands behind the ears, above the clavicle, or under the chin frequently following. When the glands within the chest become enlarged, pressure upon adjacent structures similar to that in aneurism of the aorta follows, and the venæ cavæ, esophagus, trachea, bronchi, recurrent laryngeal nerves, or other structures may be damaged. The larynx and trachea may be displaced laterally and the sternum perforated. The dyspnea, cough, dysphagia, pain, cyanosis, swelling of the neck or arms, and other results of the increasing pressure are most distressing. The enlarged abdominal glands give rise to analogous symptoms in that cavity and in the lower extremities. I have seen priapism and edema of the genital organs. Itching of the skin and bronzing may occur. A marked secondary type of anemia, frequently with eosinophilia and lymphocytosis, develops.

Pain, emaciation, asthenia, and fever add to the distress of the patient. Secondary growths may occur under the skin. The enlargement of the glands may remain localized for many months without other symptoms or with but slight fever. The type of relapsing fever in connection with this disease (Pel-Ebstein paroxysms) may be due to a secondary infection. The glands may show the evidence of an acute inflammation during the paroxysm. The spleen may be much enlarged in certain cases, in which for a time at least no involvement of the lymph glands can be detected, and some authorities class this type as "pseudoleukemia splenica."

Hodgkin's disease may at times come on very acutely, with death in the first month, but in general the course is a chronic one. If angina is present the acute cases may suggest the very acute type of lymphoid leukemia. As the disease advances the anemia becomes more apparent, and languor, dyspnea, headache, palpitation, and possibly edema of the ankles may be noted. The pressure of the lym-

phoid tissue in the pharynx may cause deafness; and neuralgic pains and even paraplegia are likewise found as the result of such pressure elsewhere. Epistaxis is an occasional symptom.

**The Blood.**—The anemia is of the secondary type and generally gradual in development. A common figure for the red cells is between two and three millions, the hemoglobin being perhaps 30 per cent. to 50 per cent. Nucleated red cells are not often present. No especial changes in the leukocytes are characteristic, but a moderate increase in the eosinophiles is occasionally reported, and more frequently a sharp increase in the proportion of small mononuclear cells, and even in the total number. A leukopenia is not infrequent, and a polynuclear leukocytosis is occasionally present. The white count is not often above 15,000 to 20,000. The diagnosis of Hodgkin's disease cannot be based upon the blood findings. Boston states that syphilis, tuberculosis, and malignant diseases so commonly present a similar picture that differentiation is impossible.

**Diagnosis.**—The most striking feature is the gradually increasing enlargement of the glands in the posterior triangle in one side of the neck with later involvement of other glands upon that side, of the opposite side, and later of the axillæ and groins, and the internal glands. If syphilis be excluded, as by the Wassermann test, and tuberculosis be eliminated, the diagnosis is reasonably assured.

**Differential Diagnosis.**—This is extremely difficult in certain cases. Lymphatic leukemia may closely resemble Hodgkin's disease, if the small cells are not greatly increased in number, for we have noted that the leukocytosis in this type of leukemia may not exceed 10,000 to 20,000, figures easily reached in pseudoleukemia. Excision and histological examination of an external gland suffice for the differentiation, since the pathology is widely different in the two diseases.

Syphilis may ordinarily be determined by the history, the presence of definitely syphilitic lesions, and the positive Wassermann reaction. Tuberculosis offers the greatest difficulty in certain cases. The enlargement of glands in this disease may be acute or chronic, and the acute general adenitis may be most deceptive. One such case died from miliary tuberculosis while undergoing investigation, before the tuberculin and Wassermann tests were available. The tu-

berculous glands more commonly appear in the anterior triangle or under the jaw, enlarge slowly, and may be limited to this locality. The glands tend to mat together, become caseous and suppurate, and search may reveal the scar of an old healed sinus. The history may point to slight enlargement for a period of years. The histological examination of an excised gland and the result of the tuberculin test are commonly decisive. In many cases pulmonary and other forms of tuberculosis may be discovered and give the clue to the diagnosis.

Sarcoma of lymph glands is not common, causes more pain than pseudoleukemic adenitis, grows more rapidly, spreads out into the tissues to a greater extent, may form metastases, and is prone to ulcerate. Lymphosarcoma, which is differentiated from the round-celled sarcoma of lymph glands by MacCullum, is very often intrathoracic or intra-abdominal in location, and pressure signs are frequently present. The histological examination offers the readiest means of differentiation. The localized adenitis from infection, and from malignant disease, should offer no difficulty if the examination be carefully conducted.

**Prognosis.**—Practically all the cases die within two or three years, not a few of the more acute type within the first few months. The possible occurrence of remissions with marked improvement as to glandular enlargement and the general condition must not be forgotten. The cases finally die of secondary tuberculosis or septic infection, from the progressive anemia and cachexia, or the pressure of the internal glands upon the vital structures.

Surgical intervention offers no relief.

## 6. ENTEROGENOUS CYANOSIS

There is a rare form of cyanosis, often associated with diarrhea or other digestive difficulties in which methemoglobin or sulphemoglobin replaces in part the hemoglobin of the blood, giving rise to the change of color indicated. It has been termed "microbic cyanosis" by Gibson and Douglas, because of the finding of a pure culture of the colon bacillus in the blood. The frequency, in the reported cases, of digestive disturbances gives credibility to the theory of Garrod

that sulphhemoglobinemia is a chronic poisoning by hydrogen sulphide, presumably absorbed from the bowel.

In the diagnosis it is first essential to eliminate the common forms of cyanosis, especially those from coal-tar derivatives, potassium chlorate, and the types associated with chronic heart disease. The use of the spectroscope is necessary to determine the nature of the change in the hemoglobin. In a case which I saw with Doctors Williams and Kleiner, the patient, a drug clerk, was able to work, but was much concerned about his ghastly color. We all believed the



FIG. 37a.—PURPURIC ERUPTION OF SCHOENLEIN'S DISEASE. (From the collection of Dr. A. J. Markley.)

condition to be due to drugs until the most rigid investigation disproved the theory. Chronic diarrhea and indigestion had existed for years. No special improvement resulted from treatment.

## 7. PURPURA

For a study of the general subject of Purpura, the reader is referred to page 30. The rheumatic form will be further considered in this connection.

### PURPURA RHEUMATICA

(*Schönlein's Disease*)

The characteristic features of the disease are the multiple arthritis, which is perhaps not rheumatic in any sense, and a multiform

eruption, typically purpuric, but perhaps more frequently erythematous or urticarial. All three of these manifestations may be present at once or at different times.

It is found chiefly in young adults, and commonly begins with a sore-throat. Malaise, moderate fever, and slight gastro-intestinal derangement are followed by the eruption, commonly first upon the legs or near the affected joints. Successive crops may develop, with great variation in the appearance of the lesions at different times. The spots show the varying colors of the usual hemorrhages into the skin during the process of absorption of the effused blood.

True rheumatic complications, notably those affecting the endocardium and the pericardium, are rare. The failure of relief by administration of the salicylates is a further argument against the rheumatic nature of the affection. Marked edema, sloughing of the uvula, persistence of the throat manifestations and relapse are mentioned by Osler as occasional features.

**Diagnosis.**—The only confusion arises in the differentiation of acute articular rheumatism when it is complicated with a purpuric eruption, commonly upon the legs. The predominance of the articular features and relative insignificance of the eruptive ones are to be noted.

## SECTION III

### DISEASES OF THE CIRCULATORY SYSTEM

#### 1. DISEASES OF THE PERICARDIUM

##### A. PERICARDITIS

Inflammation of the pericardium is commonly considered under the following classification: (a) acute fibrinous pericarditis; (b) pericarditis with effusion; (c) chronic adhesive pericarditis.

**Etiology.**—A decreasing number of cases of pericarditis are classed as of the primary or idiopathic type. More and more these are found to be due to some obscure infection or to tuberculosis. Traumatic pericarditis is not uncommon as the result of the perforation of the sac by a needle, knife, bullet or rib from without, or the passage of a sharp body through the esophagus from within.

##### SECONDARY PERICARDITIS

The great majority of the cases of pericarditis are secondary to some infectious disease, and there are few with which it may not be associated. It may arise by direct extension of an inflammatory process, the most common one being pleuro-pneumonia. Any septic or malignant process in close proximity to the heart may be the cause, and septic endocarditis or myocarditis may originate the trouble from within. The infecting organisms are probably carried by lymph vessels or blood vessels, even in certain of the cases charged to direct contiguity. Most cases of secondary pericarditis are found in association with certain diseases of well-recognized infectious character. In the order of their importance so far as we may judge these diseases are probably: rheumatic fever, which is accorded first place by all authorities; chorea; septic processes, notably puerperal fever and pyemia; pneumonia, when the question of

direct extension is ruled out, by the location of the lung troubles far from the heart; tuberculosis, and finally a group of acute infectious diseases not mentioned, of which scarlet fever is the most important. The frequency of pericarditis as a terminal event in chronic nephritis, gout and many other of the diseases which produce a chronic cachectic state is well recognized. The lowered resistance in these diseases is the great factor in the production of infections of the serous cavities at the end of life. The exudate is found sterile in many cases, but a considerable number of these prove to be tuberculous. In the absence of this cause it is assumed that the toxins of the disease cause the inflammation, as those of the gonococcus are believed to cause a gonococcic rheumatism, when no organism can be isolated. In the frankly infectious cases the streptococcus, staphylococcus and pneumococcus are most frequently found, but the colon bacillus, gonococcus and other organisms are occasionally present.

**Pathology.**—In the fibrinous form, the pericardium is covered at least in part by a thin layer of fibrin, with much injection of the serosa beneath, and occasional hemorrhages. A little cloudy exudate showing leukocytes is found in the cavity. In the more severe process the pericardial surface becomes opaque and the attached fibrinous exudate more yellow and thick, and it finally presents the shaggy condition described as *cor villosum*. The prolongations may even become attached to the parietal surface of the pericardium. The myocardium is cloudy in the ordinary cases, and more seriously degenerated in the severer ones. In pericarditis with effusion, and such effusion occurs to some extent in probably a majority of the cases, from 100 cc. to 500 cc., not infrequently much more, of sero-fibrinous liquid is present. It is generally clear, and straw-colored, with perhaps a greenish hue, in the milder cases. In the more severe ones the effusion may be reddish from fresh blood, a dirty brown or cloudy, from leukocytes and lymph fragments, and in the worst cases, purulent. The hemorrhagic effusion, as in the case of the pleura, suggest tuberculosis or malignant disease, or the cachectic condition of the aged. In hemorrhagic diseases, notably scurvy, the effusion may be very bloody. The thickened and injected pericardial

surface is covered with fibrin, or in the exudate may be found many detached fragments. In the purulent cases the friction of the two layers erodes away the surfaces coming in contact and the softened and degenerated heart muscle is exposed and breaks down.

The fibrinous exudate may, in the more chronic cases, result in the adhesion of the two layers and the obliteration of the pericardial cavity. If a considerable purulent pericardial exudate be present, the remains may become caseous and in rare cases calcium salts are deposited, so that the heart is encased in a calcareous cover. In the type called chronic adhesive mediastinopericarditis, the inflammation between the two layers of pericardium extends gradually to other tissues, the adhesions in extreme cases uniting firmly the pericardium, the chest wall, the dome of the diaphragm and all the tissues in the mediastinum. A great dense fibrous mass, including all these structures may finally be formed. The association of a chronic inflammatory process in the capsule of the liver (Pick's disease) will be considered elsewhere.

**Symptoms.**—**ACUTE FIBRINOUS FORM.**—These are often those of the primary disease, so little modified that the complication escapes detection. More cases are found in routine examination of the chest than through examination instituted because of symptoms relating to the heart. Pain or a feeling of distress about the heart with inability to lie in a comfortable position, or to get a deep breath, is often mentioned. The pain may be severe or even anginoid in character. Palpitation is not unusual. The fever of the original disease often shows no characteristic departure from its course. The pulse and the respiratory rates are raised. A slight cough may be present.

**Physical Examination.**—Inspection often shows an anxious facies, hurried respiration, and forcible, quick heart beat. On palpation the action of the heart is found to be vigorous, and occasionally irregular. The characteristic finding is that of a soft, grazing type of friction, felt commonly over the area of cardiac flatness. This is subject to very rapid modification by the changes within the pericardial sac, and may even disappear within a few hours. Auscultation demonstrates the murmur corresponding to the palpable friction,



and the auscultatory sign is often present where the palpatory one is missing. It is increased by pressure of the bell of the stethoscope against the chest wall, although this commonly increases the pain of the disease. Such pressure seems to bring the murmur nearer to the ear, but it is always a "superficial" murmur. In character, the friction murmur is a grazing or slightly rubbing sound, generally to-and-fro in rhythm and not exactly corresponding to the time of the sounds of the heart, but overlapping them. The area over which the friction is audible varies greatly, even from morning to night, but in general it may be found in the upper half of the precordial area rather than in the lower, and to the left of the sternum. It will be materially influenced by respiration. In certain cases, the sound is like the creaking of new leather, the *bruit de cuir neuf* of the French. In general it may be stated that once fairly heard this murmur is easy to recognize. In pneumonia it may be covered up by the adjacent and more obtrusive lung sounds. It is probably less often found in the region above a purulent effusion than a sero-fibrinous one, because of the lubricating tendency of the former. The action of the heart may be very much disturbed. In a severe case great rapidity and irregularity in the cardiac action suggest a serious myocardial involvement.

**Diagnosis.**—This cannot be made upon the clinical history, but audible or palpable friction in a case with a suggestive history and such symptoms as we have outlined suffices for the diagnosis.

**Differential Diagnosis.**—A double heart murmur, especially of the rough aortic type suggests a pericardial friction, but the transmission of the two different elements in the murmur, their exact timing and their individual characteristics are sufficient in most cases. The pleuropericardial murmur is heard only at points where the two serous membranes come together, is much influenced by respiration, and often ceases on cessation of respiration.

**Prognosis.**—The disease may terminate with a few trivial adhesions or serious adhesive pericarditis, but very commonly effusion occurs, in which case the murmur disappears and the cardiac area increases. If the pericarditis becomes chronic without the intervention of effusion, tuberculosis is very strongly suggested. Death

in any case is due rather to the associated disease than to the pericarditis.

### PERICARDITIS WITH EFFUSION

**Symptoms.**—These may in the beginning be exactly those described under the fibrinous form. If considerable effusion develops, the distress and dyspnea are increased, and dysphagia and cough may be troublesome. Orthopnea not infrequently appears, since the effusion not only embarrasses the cardiac action, but takes up lung space and presses upon many important organs. The circulation is so interfered with as to produce a dusky or cyanotic, anxious facies. Delirium, even severe in character, may occur. As the effusion increases, aphonia may appear from pressure upon the recurrent laryngeal nerves. A serious symptom is the paradoxical pulse, in which the mechanical effect of the effusion in hindering the filling of the heart cavities, and the entrance of the blood from the veins upon which the filling depends is manifested by a nearly complete loss of pulse, during inspiration. The nervous symptoms become prominent in grave cases. The patients are restless, sleepless, delirious and comatose, and even maniacal. Gibson describes cases which suggest delirium tremens. A very high temperature is of serious import.

**Physical Examination.**—The skin may be pale, but more commonly cyanotic, and the dusky face is fairly suggestive of the condition. The cervical veins are dilated and may pulsate. The patient may lean to the left in case of a large effusion. The apex-beat is feeble and diffuse, or imperceptible. It is generally raised to the fourth interspace, in cases where it can be found at this stage. No precordial friction is present in most cases, but if the patient lean forward the heart may come in contact with the chest wall and reproduce it.

The precordia may be more prominent than normal, especially in children. A large effusion may even depress the diaphragm, especially upon the left, and cause a prominence in the upper left abdomen. The left side of the chest is restricted in its respiratory movements.

Percussion demonstrates the flatness caused by the large collection of fluid just within the chest wall. The fluid tends to settle to the lower part of the pericardial sac, which consequently bulges, giving rise to the pyramid-shaped or pear-shaped area. The ex-

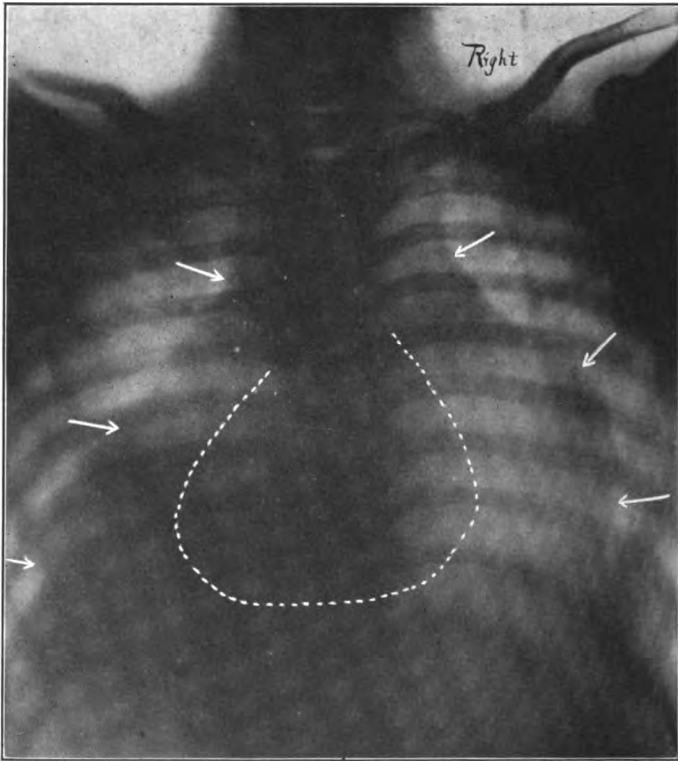


FIG. 38.—POSTERO-ANTERIOR VIEW OF THE CHEST OF A BOY 10 YEARS OLD. The arrows point to the pericardium, which is distended with effusion. Note the distension of the pericardium over the great vessels in upper part of chest. Note also the obliteration of the cardiohepatic angle. The shadow of the heart is discernible upon the plate and is indicated here by the tracing. (Dr. S. B. Childs.)

tension of the dulness beyond the point of apex-beat, or, if this cannot be felt, beyond the point of its maximum sound, thence upward above the third left costal cartilage, and on the right to beyond the sternal edge, especially at the fifth right interspace, is practically decisive as to the presence of effusion. In the last mentioned region

Rotch often found the flatness to extend 2 to 5 cm. beyond the sternal border. Traube's semilunar space may even be encroached upon by effusion. Slight dulness in the left axilla, and near the left scapula, with distant and faint bronchial respiration, due to compression of the lung by the large effusion, may be noted. A change in the area of dulness and in the signs last described upon change of position is very characteristic of pericarditis with effusion.

The auscultatory signs are of less importance, since the pericardial friction sound has commonly disappeared, and the heart sounds are much enfeebled by the presence of the effusion. In rheumatic cases especially an apical systolic murmur is often present. As the effusion disappears the pericardial friction may return.

**Course and Prognosis.**—In the secondary serofibrinous cases this depends rather upon the associated disease than upon the pericardial condition. The effusion tends toward a rapid maximum and a rapid disappearance more strongly than does the pleural exudate, and in the course of two or three days the worst stage may be over, and the effusion may have disappeared completely, perhaps with an intervening friction rub, in a week. Adhesive pericarditis results, and is a serious handicap to the heart, especially if it be burdened with the compensatory work demanded by a coincident valvular lesion. In certain cases the effusion may become subacute or even chronic. It is possible for the effusion to perforate the chest wall, but this is exceedingly rare. The immediate outlook is good in most cases, although death from gradual exhaustion or from the associated disease is not uncommon. The outlook in purulent effusion is very grave, in large part from the commonly associated general septic condition.

**Diagnosis.**—This depends largely upon the alertness of the physician in the matter of suspecting the possible incidence of a pericardial complication, the careful daily examination of the precordial area, with especial attention to any increase, and watchfulness for the development of the characteristic pyramidal area of flatness. The X-ray is of great help. Aspiration is not commonly needed as a diagnostic measure, but should be resorted to in a serious and

doubtful case, especially if the character of the fluid be seriously questioned. Perhaps the safest place is just outside the point of the apex-beat or of the loudest apical sound in the fourth or fifth space.

**Differential Diagnosis.**—For the differentiation from left-sided pleurisy with effusion see chapter upon diseases of the pleura. Mackenzie once tapped a pericardial effusion thinking it to be in the left pleural cavity, and I have seen the same mistake made.

The every day difficulty is in the differentiation from dilatation of the heart. This is not so troublesome when the case has been under observation for some time before. In dilatation, the history is that of chronic heart disease or an acute and exhausting affection, while in pericarditis with effusion there is such a history as we have outlined in the general description we have given above. Effusion occurs under febrile conditions, while the dilatation is often connected with an affection free from fever. Pain is rarely present in cardiac dilatation. The presence of a friction rub is decisive in favor of pericarditis, but it is rarely found under such conditions. The wavy heart beat of dilatation is not seen in pericarditis, and the palpable beat is often missing in this affection. The filling up of the area in the angle between the sternum and the liver (Rotch's sign) is not to be expected in dilatation. The area of flatness is more movable in pericardial effusion, and more triangular in shape. The dilated heart does not reach so high at the left edge of the sternum, unless in severe cases of mitral obstruction. The tympanitic note in the axilla could hardly be found in dilatation. With regard to the scapular area of dulness, I observed such an area in a case of transposition of the viscera with congenital heart disease, in which there was present the largest heart I have ever examined clinically, in a child of four years. It was of course upon the right side, and there was no possible question of effusion. As Osler says, the best evidence of the difficulty lies in a consideration of the excellent men who have punctured the heart in attempting to aspirate the pericardium. I have seen it done by men of much experience on several occasions. In case a pleural effusion coexists with one in the pericardium, or the lung be solidified by acute pneumonia, the

difficulties are extreme. A careful consideration of the history, the symptoms and the physical signs must be the basis for the decision, and the needle should be used, unless the decision can be reached otherwise.

### CHRONIC ADHESIVE PERICARDITIS

(*Indurative Mediastinopericarditis*)

The adhesion of the two layers of the pericardium has been discussed as an occasional result in fibrinous pericarditis, and as the

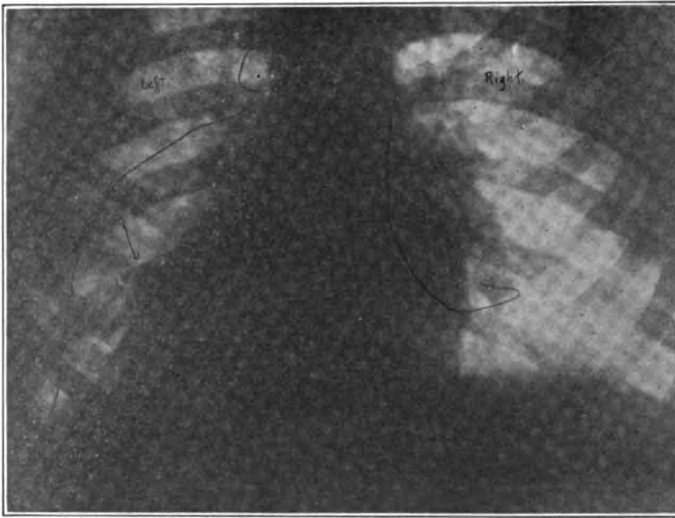


FIG. 39.—DILATED HEART. Aortic leak, mitral regurgitation. Arrow points to a pericardial adhesion. The black lines are the shadows of lead wires placed upon percussion outline of heart dullness. (Stover.)

usual result in pericarditis with effusion. In the milder cases simple adhesion of the two layers with obliteration of the cavity and a moderate interference with the cardiac function are found. In the more severe type, the general mediastinitis, already spoken of under pathology, is present. In the extreme cases, the universal contraction about the heart interferes with the orifice of the inferior vena cava, and it is in this type that the chronic inflammation affects the capsule of the liver, giving rise to a perihepatitis of extreme grade.

The involvement of the pleural sacs and the peritoneum simultaneously gives the affection described as multiple serositis, a chronic inflammation affecting each of the serous membranes described.

**Symptoms.**—In the milder form, no symptoms are present, unless arising from the faulty action of the heart, owing to associated valvular lesions or the dilatation and hypertrophy due to the interference with the free movements of the heart caused by adhesions. Even the murmurs heard are possibly relative, and due to the muscular insufficiency. When unaccountable failure in compensation occurs in a simple valvular disease, in a young and otherwise healthy individual, the most common explanation is to be found in the interference with the cardiac movement and efficiency from pericardial adhesions. Fortunately the attachments of the pericardial layers are loose enough in most cases to permit of fair movement of the heart, and no symptoms result. In the hepatic cases, the history is that of a gradually developing ascites, often with dyspnea and with so little to attract attention to any other organ than the liver that the diagnosis of ordinary cirrhosis is ordinarily made. The recurrence of the ascites after tapping, even a score of times, in a few instances, is so different from the quickly fatal result in alcoholic cirrhosis as to attract attention to the likelihood of error in case of the latter diagnosis.

**Physical Examination.**—In the milder cases nothing is apparent upon inspection, since even the systolic retraction about the region of the apex-beat fails because of the looseness of the attachments between the pericardium and the chest wall. In the severer types the enlarged cardiac area is marked out by the bulging of the chest wall, and the diffuse apex-beat is noted over a large part of this area in certain cases. The impulse is wavy and is accompanied by systolic retraction of the parietal tissues, from the firm adhesions described heretofore. These adhesions prevent dislocation of the apex-beat and of the percussion outlines in general, with change of position, whereas in the normal chest the heart is quite movable. Broadbent's sign consists of the pulling in of the chest wall because of the tug on the central tendon of the diaphragm, synchronous with the heart beat. It is seen not only in the diaphragmatic attachments, near

the seventh and eighth ribs on the left, but even better in the left dorsal region, at the region of the eleventh and twelfth ribs. This phenomenon is not pathognomonic, since it may occur in thin persons, with great hypertrophy (Tallent). The lower part of the chest is restricted in its movements, and the descent of the diaphragm is interfered with. Friedreich pointed out the emptying of the veins of the neck at the time of the filling of the heart—diastolic collapse. The diastolic rebound, felt at the apex, is of importance, but is less marked with the muscular failure of advanced disease. The area of the heart is found by percussion to be increased. This depends in part upon the increased size of the heart, but also upon the fact that it is uncovered to an abnormal extent by the retraction of the free lung border, owing to the pleural and pleuropericardial adhesions generally present. Yet the lung border may be slightly movable in some cases. The liver may show an increased area upon percussion in case of cardiac dilatation and muscular insufficiency. Auscultation may reveal much or little in accordance with the presence or absence of organic valvular disease, or the relative leakage of dilatation and muscular failure. In a young individual the presence of a systolic murmur at the apex in a heart of fair strength, would indicate a valvular lesion, but in the older cases the presumption is much in favor of the stretching of the mitral ring, with relative insufficiency. A presystolic murmur is occasionally present in adherent pericarditis, without the presence post mortem of the signs of mitral stenosis. As in all cases of exhausted heart, tricuspid leakage may be present.

**Diagnosis.**—With the group of symptoms described and the presence of ascites and not rarely of double pleural effusion, the diagnosis of Pick's disease may be made. The local signs referring to the heart alone signify an ordinary adhesive pericarditis.

**Prognosis.**—The cases with simple adhesions, not extending into the tissues around the heart, cannot be diagnosed during life, and have presumably a normal expectancy of life. If this fails on account of the heart it is to be interpreted as meaning that there was more than simple adhesion present. In the more severe type death occurs as in ordinary valvular disease, from final dilatation and



weakening of the heart muscle. In the indurative type the outlook is bad, but the subject may live some years under favorable circumstances. The prospect of relief from Brauer's operation is only a fair one, and this only in selected cases.

### OTHER AFFECTIONS OF THE PERICARDIUM

**Hydropericardium.**—Hydropericardium occurs in cases of general anasarca, but less frequently than ascites and hydrothorax. In case both pleural cavities contain fluid, it is of difficult diagnosis. Dropsical accumulation in the pericardium alone is rare, but is said to occur after scarlet fever.

**Hemopericardium.**—This term is applied to the effusion of blood into the sac, generally from rupture of the cardiac wall, wound of the wall or rupture of an aortic aneurism within the pericardial attachments. If the hemorrhage continues the intrapericardial pressure becomes so great as to prevent diastole, and death results. The condition must not be confused with pericarditis with bloody exudate described elsewhere.

**Pneumopericardium.**—Pneumopericardium is a rare condition, due to the entrance of air or gas into the sac, either from external trauma, perforation of the sac by malignant disease or an air- or gas-containing viscus (stomach, esophagus) or possibly from the presence of gas bacilli. The "splashing sound of the mill" is almost pathognomonic. The presence of air of course causes the flatness of the precordial area to give way to tympany.

## 2. AFFECTIONS OF THE MYOCARDIUM

### A. HYPERTROPHY

The muscle walls of the heart increase in thickness and in strength, if properly nourished in accordance with the demands made upon them for work. If more blood is to be moved for any reason, the cavities dilate, and the muscle wall hypertrophies to meet the increased work necessary to empty the cavity. As a rule, then, the

hypertrophied heart is one with enlarged cavities, but in case the work is increased solely through the increase of pressure against which the organ must labor, the cavities may remain of normal size, but with thickened walls. Various valve lesions necessitate dilatation and hypertrophy of certain cavities called upon to make up by compensation for the leakage or obstruction, so that hypertrophy of the heart does not necessarily imply a universal hypertrophy, which in fact is less common than overgrowth of the walls of particular cavities. Hypertrophy of the heart should be looked upon as merely the manifestation of that process which regulates the size and strength of a muscle according to the work it is called upon to do. The materially heavier heart of the athlete, as compared with that of a man unaccustomed to severe muscular exercise, and the response in undertaking a course of training, in the development of a greater heart area and corresponding heart strength, have the most direct bearing in a consideration of hypertrophy from disease. Yet the heart does only the amount of work actually required by the immediate conditions. It is in health able to do for a short time about thirteen times the amount of work necessary in a condition of rest.

Hypertrophy of the left ventricle may result from: (a) overwork. It is common in athletes, laborers in certain severe occupations, and in mountaineers. It is to be regarded more as a physiological than a pathological development. (b) Conditions outside the heart increasing the amount of work done. Arteriosclerosis is the most common example, with the increased pressure associated with arterial and perhaps concomitant renal disease. The aorta and splanchnic vessels are commonly most affected in cases with marked hypertrophy of the left ventricle. The irritation and response by contraction of the smaller arterioles from certain substances in the blood in the first hours, even, of an acute nephritis may raise the pressure and lead to a demonstrable cardiac hypertrophy before arterial changes of any degree can be found. (c) Conditions within the heart, notably aortic obstruction and insufficiency, and mitral insufficiency. (d) Pericardial adhesions increasing unduly the work of the heart because of the "drag" associated. Myocarditis may interfere with the "tim-

ing" of the different cavities of the heart in such a way as to make it work at a disadvantage, with consequent hypertrophy. (e) A failure of proper regulation of the heart, from nervous derangement, such that it beats too rapidly and forcibly, and the muscular tissues respond by hypertrophy. The best example is the large heart of nervous palpitation, whether induced by tobacco, coffee, alcohol, or the cardiac stimulation of exophthalmic goiter. In the "soldier's heart" there is added the muscular work of severe campaigns, and in the "beer heart" the necessity for moving a large amount of fluid, so that the nervous element cannot be looked upon as the sole cause.

In the case of the right ventricle, the chief cause is found in increased work because of higher pressure in the pulmonary system. It is due to backing up of blood in the lungs as a consequence of mitral disease, or to the emphysematous or fibroid changes in the lung which obliterate a sufficient number of capillaries to narrow materially the blood path. Valvular lesions are of less frequency, but operate in the same manner as upon the left side. In the case of the left auricle the hypertrophy is always associated with dilatation, and occurs as a result of regurgitation, and especially stenosis at the mitral orifice. In the right auricle the increase is found more frequently in association with that of the ventricle than as a result of auriculoventricular valvular disease.

**Pathology.**—The muscle fibers are increased in volume, and the wall of the affected cavity is thickened, even to the extent of two or three times the normal measurement.

**Symptoms.**—Excepting for the "pounding" in the nervous type described, the symptoms of hypertrophy of the heart do not appear until this hypertrophy fails. It is a conservative process, necessary to a continuance of a fair circulation.

**Physical Examination.**—Hypertrophy of the left ventricle is the variety which most commonly attracts attention and is most frequently diagnosed. The heart is enlarged, oftentimes showing the apex-beat far outside the mid-clavicular line and lower than normal. The action of the heart is heaving and forcible, the aortic sound is accentuated, and the first sound has a booming quality, or even a "clink," especially common in conditions of unusually high blood

pressure. The pulse is full, and a greater or less degree of increased vascular pressure is commonly present.

Hypertrophy of the right ventricle leads to an increase in the precordial area, especially to the right, although this may be undemonstrable because of the associated lung disease which has led to the hypertrophy. The apex-beat is less sharply defined than normal because of the covering of the left ventricle by the enlarged right cavity. Pulsation in the epigastrium is a very frequent and reliable sign of the condition. The pulmonary second sound is commonly accentuated. The systolic venous pulsation in the neck is a sign of failure of the tricuspid valve, and not of the hypertrophy of the ventricle proper.

Hypertrophy of the left auricle is inferred from the associated conditions rather than physically demonstrated. If dilatation occurs, the Röntgen-ray plate shows it distinctly. Hypertrophy of the right auricle may be demonstrated by careful percussion in certain cases, the increase in area lying to the right of the sternum below the third rib. This is more commonly to be found after dilatation has occurred. An auricular venous pulse wave may be present in the neck. A systolic venous pulse signifies that the tricuspid valve has become incompetent.

**Diagnosis.**—The hypertrophy of the walls of the different cavities of the heart is so intimately associated with various valvular lesions, and so commonly found with varying degrees of dilatation, that the attempt at differentiation, further than indicated above, would not be profitable.

## B. DILATATION

An acute dilatation of the cavities of the heart may be demonstrated with the Röntgen ray after almost any severe exertion, and doubtless occurs in everyone at certain times. Immediate restoration to normal is the rule, and the process is not pathological, nor is it associated with hypertrophy of the heart. As Mackenzie states, in order that dilatation of the heart may take place it is necessary as a prerequisite to assume a deficiency in tone of the cardiac muscle. This "tone" is the element in cardiac physiology which keeps the

muscle in "a position short of extreme relaxation." The auricle might be overpowered by the powerful ventricle if the valve gave way, but in general the features of which we shall next speak, which produce dilatation, do not begin to act until the deficiency in tone is pronounced.

The causes of permanent dilatation of the heart have been in part discussed under hypertrophy of that organ, since the latter is a necessary accompaniment of the former in the beginning, although the dilatation assumes the chief rôle as the heart finally fails. The

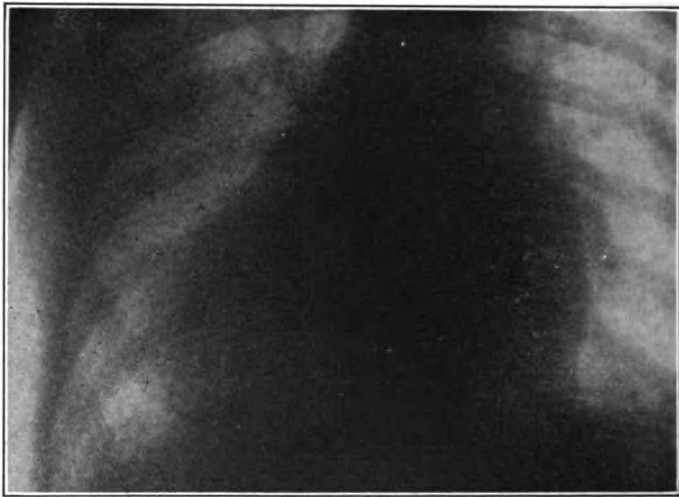


FIG. 40.—DILATED THINNED HEART MUSCLE. (Dr. G. H. Stover.)

earlier dilatation is due to the increased blood pressure within the particular cavity affected, while the latter type is due to the eventual failure in the strength of the muscle wall. The dilatation due to the increased blood pressure is in general a conservative process, the increased power of the heart from the resulting hypertrophy being necessary because of the work to be done. Without dilatation and hypertrophy the heart of mitral regurgitation would shortly be entirely incompetent, for example, or the heart of the young man training for a race would be unable to sustain him through the ordeal because of lack of capacity for handling the increased traffic through the circulatory organs. The gradually induced dilatation of the na-

ture described leads to no serious results. Entirely different is the acute dilatation from the severe exertion in the untrained individual, or more frequently in the one with limited heart capacity, due to arteriosclerosis or other cause of beginning degeneration. In such subjects, overexertion, of which the most common type is probably that involved in hill- and especially mountain-climbing, leads to acute dilatation, shortness of breath, with finally utter inability to make further effort. The cardiac area increases widely, and may not be again entirely restored. Very moderate exertion thereafter brings on at once symptoms of recurrence, and the individual is "broken winded," as the term is applied amongst horsemen. A due regard to the marked oppression and dyspnea, felt at the time when we may conclude that the "safety valve action" of the tricuspid interferes so markedly with the pulmonary circulation, would save many a man after middle age and a certain number before that time from permanent cardiac damage. It is unnecessary to speak further of the dilatation occurring in connection with the different valvular lesions, since the matter has been discussed under hypertrophy. In many chronic diseases of the heart with feeble pulse, death occurs from eventual cerebral thrombosis, or other lesion directly dependent upon the insufficient blood pressure. Of the causes which weaken the resistance of the muscular wall, and permit dilatation to occur, even with a normal or possibly subnormal pressure, we shall speak in the sections upon the degenerative diseases of the myocardium.

### C. CARDIAC INSUFFICIENCY

**Acute Form.**—The causes of acute cardiac insufficiency as given by Gibson are as follows: (a) wounds of the heart, (b) spontaneous rupture or rupture of valves, (c) hemopericardium of rapid production, (d) overexertion, (e) pressure of large amount of air in the heart, (f) heart thrombi, (g) sudden obliteration of a large section of the lung arteries, (h) sudden interference with coronary circulation, (i) mechanical interference with the heart as in asphyxia, (j) result of infections, (k) poisons, (l) interference with the nerves of the heart, especially the vagus.

These various topics are all considered in the sections under which they naturally fall.

**Chronic Cardiac Insufficiency.**—The table of causes given below is modified from Gibson:

(a) Lesions of the heart muscle. (*See Section on Diseases of the Myocardium.*)

(b) Lesions in the valves. (*See Valvular Diseases.*)

(c) Lesions peripheral to the heart itself. In the case of the left ventricle, a continuance with gradual progress of the usual causes of increased blood pressure, of which the chief ones are interstitial nephritis, general arteriosclerosis, with especial involvement of the splanchnic vessels, and atheroma of the aorta, eventually exhausts the reserve power of the ventricle and it dilates. Upon the right side emphysema and chronic fibroid disease of the lung finally produce a similar result.

(d) Interference with the proper movement of the heart, as by adherent pericarditis.

(e) Goiter. By obstruction of the air passages an increased expiratory pressure is produced within the lung, is communicated to the vessels, and results in increased work on the part of the right heart. The further developments are similar to those in emphysema, as described above.

(f) Excessive stimulation of the heart, as by overwork, atheroma of the aorta, general arteriosclerosis, and muscular failure of the heart itself are important features here.

(g) The action of poisons, alcohol, tea, coffee, and tobacco especially. It is thought by physicians in the mining regions that a severe type of chronic myocarditis at times results from cyanid poisoning in those employed in the works where gold is recovered by the use of potassium cyanid.

**Pathology.**—Acute degeneration of the myocardium is responsible for many cases of cardiac insufficiency. In the acute infectious fevers a parenchymatous degeneration of the heart muscle takes place under the influence of the toxins of the disease. Diphtheria, typhoid fever, small-pox, grippe, scarlet fever, and rheumatic fever are the usual causes. It is to be noted that the symptoms may not become

manifest until the infectious process has subsided. The toxemias of pregnancy and pyemia are infrequent causes of this type of degeneration. Acute degeneration follows plugging of the coronary arteries by an embolus or by thrombosis, the former occasionally septic in nature and much more serious in effect. The more acute the blocking of the circulation by the thrombus the more serious is the degeneration. In the area of anemic necrosis produced, rupture of the heart wall may occur. In the acute interstitial type of myocarditis, in which the connective tissue of the heart wall is swollen and infiltrated with lymphoid cells, the muscle fibers undergo an acute degeneration. In septic embolism abscesses arise from the infarctions produced, often multiple. If an abscess perforates the heart wall it produces the so-called acute ulcer of the heart.

Sudden death is not infrequent as the result of the slowly developing coronary obstruction, as by atheroma, with a gradual fibrotic change from insufficient nutrition. If the remaining lumen become occluded death commonly occurs, since the collateral circulation through the branches of the other coronary artery and Thebesian vessels is rarely sufficient. The fibrous area may stretch, producing an aneurism of the heart, or may even rupture, as in the acute type described. A chronic myocardial degeneration is a fairly frequent complication of uterine fibroids, of chronic gall-stone disease, goiter, and many other conditions.

**FATTY DEGENERATION.**—The heart muscle looks yellowish-brown and patchy and offers little resistance. Fat droplets replace the degenerated muscle fibers, and eventually a fibroid patch may indicate the point of the previous fatty change. This type of degeneration is common in any condition in which marked nutritional failure is found, as in old age, pernicious anemia, and after prolonged acute illnesses. It is not infrequent in the hypertrophied heart-wall after the nutrition suffers from the advance in the cardiac disease. The constant activity of the heart muscle renders it excessively sensitive to any interference with its nutrition.

**FATTY HEART.**—In the condition of fatty overgrowth, the heart is not only covered with fat, but it infiltrates the wall between the fibers of the muscle and impairs the ability of the heart to bear sus-



tained exertion. The necessity of the movement of the epicardial fat with every contraction of the heart is in itself a considerable burden. Fatty degeneration of some of the muscular fibers in the heart wall may be found.

**FRAGMENTATION AND SEGMENTATION.**—In the death agony the muscle fibers may rupture at the region of the nucleus and separate at the line of the cement substance. Both processes may be present in the same specimen. Other types of degeneration may be coincident. Although the condition undoubtedly occurs in disease, it is unrecognizable clinically. The brown atrophy seen in advanced cases of valvular disease may be another cause of the giving way of the heart muscle when under stress.

**Symptoms of Cardiac Insufficiency.**—Clinically we find failure of the function of the heart whatever the type of degeneration in the cardiac muscle. It is the inability of the muscle to perform its work, not the nature of the lesion, that produces the symptoms, although the latter vary sufficiently to give fair ground for some distinction between the different types. In the acute parenchymatous form the pulse is commonly increased in frequency, and of low tension, with a tendency to become irregular and more rapid upon exertion. Somnolence and pallor are occasionally noted at this time. Vomiting and slight precordial pain are common. Slight dilatation, weakened sounds, and feebleness of the pulse may be found upon examination, and a relative mitral regurgitation is often present. Under sudden exertion, dilatation and death may quickly follow. Mackenzie states that in this type the "irregularity of the heart is due to dropping out of ventricular systoles because of damage to the auriculoventricular bundle."

In the acute interstitial form malignant endocarditis is closely simulated, the infarcts over the body being due to the rupture of a parietal abscess into one of the heart cavities. In chronic myocarditis we may speak of a latent group, in which sudden death occurs without a history of any symptoms, and a cardiac group, in which the heart symptoms have been conspicuous. In the former the insidious myocardial degeneration may have reached Kronecker's center; or the dilated and hypertrophied heart has suffered a gradual

change such that a sudden rise of blood pressure, as from emotion or from exertion, stops the heart in diastole. Doubtless most or all of the latent cases would have shown dilatation or hypertrophy or both, an increased blood pressure, a quickened pulse, and dyspnea upon exertion.

In the second group are the patients with recognized hypertrophy of the heart, increased tension, a trace of albumin, and a few hyaline and granular casts, dizziness, dyspnea, and some digestive distress. On examination, the hardening of the arteries, the increased area of the heart, accentuated aortic sound, possibly soft apical systolic murmur, and puffiness of the ankles are significant of the myocardial disease. In the distinctly renal cases, a higher blood tension and more striking urinary signs are noted, and the outcome is likely to be a greatly dilated heart, low blood tension, mitral and tricuspid leakage, and general dropsy. The heart action becomes very rapid and irregular at the end, often with gallop rhythm. Other cases present the Stokes-Adams syndrome, with bradycardia, vertigo, syncope, and convulsive attacks. Death probably takes place as a result of a complete heart block.

Anginal attacks are not infrequent in the subjects of chronic myocarditis. In the typical cases the arteries are hardened, some hypertrophy is evident, the aortic sound is accentuated, and the pressure is increased. The affection of the myocardium is commonly secondary to sclerosis of the coronary arteries. The attacks of pain may be the sole obstacle to fairly rapid walking or other exercise, although many have severe dyspnea aside from the pain. In general the indications of cardiac insufficiency caused by the conditions we have been considering may be pain, dyspnea, sleeplessness, drowsiness, pronounced mental symptoms, Cheyne-Stokes respiration, Stokes-Adams syndrome, cyanosis, pallor, edema, albumin and tube casts in the urine, evidence of hypertrophy and dilatation of the heart, and appearance of bradycardia, arrhythmia, gallop rhythm and various murmurs, and an indigestion due to the passive congestion in the liver, stomach, and other digestive organs after the heart power fails. Yet in certain cases none of these symptoms is recognized, since the patient dies suddenly, while others have them in various groupings. Of these the

anginal and Stokes-Adams types are the most distressing and the most frequently fatal.

**Prognosis.**—Nothing is more uncertain than a prognosis in cases of this group, since we cannot know how serious is the myocardial degeneration. The prognosis in all is bad, but better in some than in others. Favorable features are predominance of the signs of hypertrophy over those of dilatation, absence of arrhythmia, tachycardia, gallop rhythm, and of anginal attacks.

### 3. FUNCTIONAL DISEASES OF THE HEART

#### A. PALPITATION

By this term is meant such disorder of the heart action that the subject becomes conscious of it. This action is generally rapid and forcible, and in many cases is irregular as well. The causes of the latter feature will be discussed later. It should be understood that a much disordered heart action may be present, but not perceptible to the patient, while on the other hand the patient complains of distress from this source, but no trouble can be found by the physician.

**Etiology.**—It is a disease more especially of females, and particularly those with an excitable nervous system. The hysterical and neurasthenic are especially subject to functional heart disorders. Given this preliminary condition, almost anything may act as the exciting cause. Attacks at the menstrual period are common, and in general the time of puberty and the climacteric are especially favorable for its development. Worry, anxiety, domestic troubles, fright, loss of sleep, the consequent weakening of the general strength, and frequently the use of tea and coffee to overcome the feeling of exhaustion so common in these subjects, may be the exciting causes. In men tobacco is probably the most frequent single cause. It must not be overlooked in women, as I have seen attacks in them due to cigarette smoking, pipe smoking, the chewing of tobacco, and the use of snuff. The three latter methods of use are frequently wholly unknown even to intimate friends. Alcohol is a common cause in both sexes. Slight causes may bring on an attack of palpitation after

the exhaustion of acute disease, especially typhoid. A single cigar in convalescence may initiate an attack. In the "soldier's heart" of DaCosta, the attacks of violent over-action lead to hypertrophy of the heart, the neurotic over-action being a prominent feature in producing it. His cases were generally connected with the excitement and over-exertion of the soldier's life, and with diarrhea, but in the rather unusual cases presenting at the present time neither over-work nor digestive disturbances may be traceable, yet the hypertrophy from overaction is well marked.

A most important cause of palpitation is indigestion, especially the type associated with the presence of gas in the stomach. The heart condition appeals so much more strongly to the patient's mind than the less obtrusive gastric condition that he commonly feels sure that he has organic cardiac disease.

**Symptoms.**—The patient complains of violent action of the heart, generally sudden in origin, and of a faint, distressing feeling in most instances. The throbbing may be felt in the arteries as well as in the heart itself, and may cause perceptible vibrations in the whole body. The pulse commonly rises to a frequency of 120 or 150 or even more at times. The frequent statements that the "heart stops and turns over" or "flops" apparently relate to the distress which the patient perceives at the time of the omission of the beat, or of an extra systole. A heightened sense of appreciation of the cardiac movements is the chief feature in this form of trouble. Young men of athletic build who smoke too freely sometimes stop the attack instantly by stooping and flexing all four limbs sharply, apparently throwing a temporary overload onto the heart, which causes it to return to its regular rhythm.

**Physical Examination.**—In simple palpitation the heart is of normal area, and if examined when not under excitement is found to have perfectly regular and normal sounds. During the attack the sounds are sharp and strong, the basic valvular sounds often accentuated. The pounding of the heart against the chest wall is very obvious. Various functional murmurs may be present, but their functional nature may be established by the examination of the heart later, when normal size, normal valvular action, soft arteries and nor-

mal blood pressure and urine are found. A low blood pressure with marked vascular relaxation has been found by Mackenzie. The attacks may pass off in a few minutes, but in the neurasthenic and hysterical may last for a considerable time, and recur several times in a day. The outlook is good as to life, but the immediate prognosis depends rather upon the underlying cause. Neurotic young men who have once established the attacks by the overuse of tobacco commonly suffer for years, even after giving up the habit.

### B. ARHYTHMIA

The modern conception of irregular rhythm in the heart's action is based upon the myogenic theory of that action. Gaskell states the functions of the heart muscle to be five in number, as follows:

"(1) The power of producing a stimulus which can excite the heart to contract, stimulus production;

"(2) The power of being able to receive a stimulus, excitability;

"(3) The power of conveying a stimulus from fiber to fiber, conductivity;

"(4) The power of contracting when stimulated, contractility;

"(5) The power to maintain a certain amount of contraction even when the active movement has ceased, tonicity."

The following classification of the irregularities of the heart is abbreviated from Mackenzie:

(1) Sinus Irregularities.—This form of irregularity is characterized by a varying length of the cardiac cycle, mainly of the diastolic portion, the pulse beats being always of equal size or nearly equal size, and presenting no "imperfect systoles" or "missed beats." The variation usually corresponds with certain phases of respiration.

(2) Extrasystoles.—Here an auricular or ventricular systole, or both together, may start prematurely and independently of the sinus rhythm. They occur occasionally in an otherwise regular heart. A premature beat of the radial pulse is felt, followed by a long pause, or there may be simply a long pause (intermittent pulse).

(3) Nodal Rhythm.—The starting place of the contraction is no longer at the sinus, but in some part lower down, where the auricle

and ventricle are stimulated to contract for the most part simultaneously. Beats of varying size follow one another at varying intervals; sometimes the irregularity is extreme, sometimes scarcely perceptible, but careful analysis will usually show variations in the length of the cardiac cycle. This irregularity is usually associated with marked diminution of the heart's power.

(4) **Irregularities Due to Failure of the Conducting Power of the Primitive Bundle.**—This is due to the ventricular systole dropping out in consequence of the stimulus for contraction not reaching the ventricle.

(5) **Depression of Contractility (pulsus alternans).**—In irregularities due to the failure of the contractile power of the ventricle the beats are usually regular in rhythm, varying in strength only.

(1) **Sinus Irregularities.**—These disturbances are chiefly due to the action of the vagus, and are analogous to the irregularities often found normally in the dog, and which disappear on section of the vagus. The pulse rate varies especially with inspiration, but the beats are equal, and a tracing shows that auricle and ventricle beat normally and in proper rhythm. Ordinarily there are no symptoms in this type of irregularity. If found during a fainting attack an undue importance may be attached to it. If the delay between the beats be very long, giddiness, and even temporary loss of consciousness may result.

**PROGNOSIS.**—It is of little importance. The irregularity of the pulse of this type in tuberculous meningitis is of value in the diagnosis, but not in the prognosis. Mackenzie states that this phenomenon is even of favorable import after, for instance, rheumatic fever, since it is not found if the muscle of the heart be exhausted.

(2) **Extrasystole.**—This is the most frequent type of arrhythmia. It is recognized by the occurrence of a premature beat followed by an abnormally long pause. In case the ventricular contraction is weak the beat is not perceptible to the finger, the result then passing for intermission, so far as interpretation of the pulse action alone is concerned. The sphygmograph reveals the true situation. In case such extra systoles appear regularly and fail to give a pulse beat, the diagnosis may be bradycardia, but the stethoscope shows the heart

beat at the time of the missed pulse beat. The extra systoles are believed to be auricular or ventricular in origin, or to arise from the auriculoventricular node. The so-called "compensatory pause" after the extra systole is accounted for by the temporary exhaustion of excitability of the ventricle so that it fails to respond to the next stimulus, and hence does not contract until the next succeeding one. The patient may be conscious of a strong beat following the missed one. This is the type of irregularity seen most commonly in practice, and may occur in many conditions both of health and disease. It may last through life without any evidence of harm. In the absence of the evidences of organic disease the condition should not be taken too seriously. In old subjects the trouble is commonly due to the effects of arteriosclerosis upon the coronary arteries. In dyspeptics and neurotic individuals the condition is not uncommon, and its frequent occurrence in young healthy individuals should be noted.

(3) **Nodal Rhythm** (Continuous Irregularity of the Heart).—

"The great majority of cases of nodal rhythm are found among those who have suffered from rheumatic affection of the heart, or cardiosclerosis. In rheumatic hearts there are often deposits of cells scattered through the heart, which ultimately cicatrize. These deposits when on or near the auriculoventricular bundle impair its function, and this is frequently recognized in early cases by the delay which occurs between the contraction of the auricle and ventricle, from the retarded transmission of the impulse through the connecting fibers. A later result may be obtained when the cicatrization irritates the bundle, and renders it more excitable than the sinus. In accordance with the law that the contraction starts at the most excitable part of the primitive tissue, the contraction of the heart then originates in this more irritable part. Somewhat analogous changes follow in cardiosclerosis and in degeneration of the coronary arteries. In certain typical cases of this irregularity Keith has found the artery supplying the bundle affected with marked arteriosclerosis, and with an invasion of fibrous tissue in and around the auriculoventricular node and bundle.

"In these cases the increased irritability of the auriculovent-

tricular node and bundle seem to lead to the inception of the abnormal rhythm." \*

This is a condition in which the so-called "fibrillation of the heart" occurs. The ventricle contracts first, interrupts the action of the auricle so that the latter becomes dilated, and, especially when diseased, speedily loses its reserve force. Clinically fibrillation of the heart is most often seen in advanced mitral stenosis with rapid pulse, the extreme irregularity of this affection being due to this cause. Rapid and almost futile contraction of the auricle may be proven by the tracing, but the presystolic murmur disappears, since the auricle does not contract normally and produce it. The veins in the neck show a rapid pulsation. If the condition continues dilatation of the heart, cyanosis, edema and death ensue. A notable feature in these cases is the "fluttering" feeling in the cardiac region, different from any usual palpitation.

**PROGNOSIS.**—If the heart beat is not materially increased in frequency the younger patients with nodal rhythm may go on with fair comfort for years and do fairly heavy work. The outlook depends upon how well the heart supports the new order of things. In general if there be but little distress the outlook is good. Even with some edema, the patient may still recover and live some time with occasional breakdowns. Cases with persistent attacks and symptoms of heart failure turn out badly.

(4) **Heart-block** (Stokes-Adams Syndrome).—In this condition the normal stimulus for cardiac contraction is unable to pass from the auriculoventricular node through the auriculoventricular bundle, because of its being interrupted at the point of the muscular connection between the auricular and ventricular chambers (Gaskell's bridge). The ventricles immediately begin to contract independently from their inherent automatic rhythmicity but at a rate much slower than that of the auricles, which normally "time" the heart. The stimulus may be delayed only, or blocked intermittently or permanently according to the nature of the lesion. Experimentally it is possible to make the rate between the auricle and ventricle two, three or four to one, in accordance with the degree of compression

\* Quoted from Mackenzie.



upon the fibers of the bridge. If then the conductivity of the bundle be depressed so that the stimulus frequently fails to cross, as when repeated stimuli from the auricle have exhausted it temporarily, missed beats will result. If the block be complete, independent ventricular rhythm is established. At times many seconds may pass without a ventricular contraction, and the failure of supply of blood to the brain gives rise to dizziness and syncope. If it lasts beyond twenty seconds on the average, the muscular twitchings of the Stokes-Adams disease occur.

**Symptoms.**—The manifestations of heart-block are the slow ventricular rhythm and occasionally slow pulse beat, 20 to 40 per minute, but even below 20 occasionally; the occurrence of attacks of giddiness, syncope and convulsive phenomena, when the ventricles stop for a time; and the visible venous pulse in the neck from 2 to 4 times as frequent as the pulse beat, and of auricular origin. The syncopal and convulsive attacks may occur without cessation of the pulse, but in these cases it is found to slow from perhaps 30 to 6 or 10 per minute. Cheyne-Stokes respiration may appear. A feature which should be mentioned is that, although the vagi still exert control over the auricles, so that exertion, excitement, etc., cause them to beat more rapidly, the ventricles are not affected.

The disease is often of syphilitic origin in the young, but in the old is commonly associated with degenerative changes of arteriosclerosis. A functional type probably exists, since in certain neurotic individuals the symptoms have been present without any lesions after death.

In all cases excepting the last class the outlook is decidedly serious, although the syphilitic type may recover under specific treatment. The patients of the arteriosclerotic type may live for years, even with a dozen attacks a day.

### C. TACHYCARDIA

The name refers to pathological frequency of the heart's action. The number of beats rises from normal to 100 or even 200 per minute, the extra beats taking time from the diastole rather than chang-

ing the time of the heart's cycle otherwise. Thus the long pause is shortened in correspondence with the increase in rapidity of the action. In the very rapid action embryocardia is to be noted, the first and second sounds being indistinguishable.

**Etiology.**—Many cases are of reflex origin, as from irritation of tuberculous disease within the chest, or from the generative organs in women. Nothnagel believed the disease to arise from temporary inaction of the vagus center, and in certain cases there may be other bulbar symptoms, as spasm of the glottis, or stridulous respiration, to confirm such a hypothesis. It is well recognized that lesions about the vagus center, or causing interference with the nerve in its passage, may be associated with tachycardia.

**Paroxysmal Tachycardia.**—Mackenzie finds some cases of paroxysmal tachycardia to be of auricular origin, extra systoles starting from this point in many cardiac cases, and tachycardial attacks having the same origin. More commonly the origin is in an abnormal irritability of the auriculoventricular bundle or node, due to malnutrition as from arterial disease. On account of its excessive irritability it starts the rhythm of the heart. Definite fibrosis of the bundle of His has been repeatedly demonstrated in these instances of nodal tachycardia. The third variety is not so well established, but is believed to originate from an irritable focus in the ventricular muscle, doubtless in an extension of the ventricular portion of the auriculoventricular bundle. Some type of nutritional damage, temporary or permanent, to the portion of the heart substance involved, has been found in many autopsies. The auricular type gives the greatest number of heart beats per minute.

**Symptoms.**—The pulse rate rises generally to 150 to 200 beats per minute, oftentimes after a preliminary intimation of the attack through the occasional occurrence of an extra systole, or an intermission perceptible to the patient. Oppression in the precordial region and distress in the left arm are not uncommon. Most patients feel compelled to lie down, but some may continue their occupation. Cyanosis, or more commonly pallor, may be noted. In many patients there is a feeling of great anxiety and distress. One physician under my observation has had the attacks for many years, apparently with-

out harm, but dreads them as many patients do the recurrence of anginal attacks. The disease may persist for half a lifetime. The individual attack lasts from a few minutes to a few hours, or even a week or a month, ceasing as suddenly as it began. In the latter cases the signs of myocardial failure occur, with dilatation of the heart, edema, cyanosis, and albuminuria. Death eventually occurs in such an attack in a considerable number of cases. The prognosis is therefore always to be a guarded one.

#### D. BRADYCARDIA

Abnormally slow cardiac action should not be confused with abnormally slow pulse beat. In normal bradycardia all the chambers of the heart participate in the slow action and the pulse beat corresponds. In the second class, as given by Mackenzie, a slow pulse rate is due to failure of certain ventricular contractions to carry the pulse to the wrist; the third class comprises the cases in which the nodal rhythm is present, the auricle either having ceased to beat, or beating synchronously with the ventricle. In the fourth class, the auricular beat is normal, but the ventricle beats slowly because of the blocking of the stimulus between auricle and ventricle (heart-block). In the fifth class the vagus produces complete standstill of the heart for a brief period.

In many individuals the normal pulse is sixty, fifty or even forty per minute. The slow pulse of age, of the inanition of starvation and of the puerperal period may be noted.

Pathologically bradycardia is most frequently seen after acute febrile disease (post-febrile bradycardia). Typhoid, pneumonia and acute rheumatism are the diseases with which it is most often associated. I have seen a marked instance after facial erysipelas. The type seen after diphtheria should be included in this class, only in cases in which vagus neuritis and acute myocardial denegeration may be excluded. In the course of many chronic diseases it may be present, notably in nephritis and uremia, in cancer or other chronic digestive diseases, in emphysema, severe anemia, diabetes, and in certain insanities. In jaundice it is commonly due to the slowing

effect of the absorbed bile upon the heart. In organic heart disease, especially fatty degeneration, it is not infrequent. The cause in many cases lies in the degenerative effect produced upon the cardiac structures regulating the rhythm, as upon the bundle of His. Valvular disease is frequently associated. The bradycardia of digitalis is common, and must be excluded. Thus I saw a patient after the crisis in acute pneumonia with a pulse of less than 40, the condition being called a bradycardia. He had been taking half-ounce doses of the infusion of digitalis, which had acted with its customary force after the subsidence of the fever. The slowing effect of brain tumors, apoplexy and other cerebral conditions is well recognized.

#### 4. ENDOCARDITIS

This occurs in two forms, the acute, common in connection with acute diseases and characterized by the development of vegetations and damage to the structure of the valve, and the chronic form, a sclerotic change resulting from the acute, or occurring as a result of arteriosclerosis, with thickening and deformity of the valves. The endocardium of the remainder of the heart is usually not affected.

##### A. ACUTE ENDOCARDITIS

As a matter of convenience, cases are classified as simple and malignant, although no sharp dividing line exists.

**Etiology.**—For practical purposes, we may consider that endocarditis is a process secondary to some variety of infection. By far the most frequent association is with acute rheumatism. Chorea, scarlet fever, tonsillitis, small-pox, measles and pneumonia may give rise to acute endocarditis of the simple form, while the septic diseases, pneumonia and gonorrhea, are the most frequent causes of the malignant type. Many cases occur, however, upon valves already damaged by previous chronic sclerotic changes. A certain number of malignant cases originate, so far as we know, as a primary affection of the valves.

The organisms causing the endocarditic process are the pneumo-

coccus, staphylococcus pyogenes aureus, streptococcus, gonococcus, typhoid bacillus, Klebs-Löffler bacillus, and occasionally the B. coli, the meningococcus and the tubercle bacillus. In the mild subacute type of malignant endocarditis described by Libman (subacute bacterial endocarditis), the organism is the streptococcus viridans, though Rosenow states that the cocci are derived from the pneumococcus. In certain cases it is thought that endocarditis originates from the action of the toxins, since no infection can be found upon the valve.

**Pathology.**—The changes in endocarditis are upon that portion of the endocardium where the most wear and tear comes, namely, upon the valves, and especially upon those carrying the heaviest load, and upon those parts of the valves suffering most from the trauma of constant use, that is, upon the edges at the points of contact. The valves upon the right side in the fetus and the left side in the child and adult thus suffer more than the others in their respective conditions. Although in certain types of endocarditis, notably in the chronic infectious form, the lesions affect the mural endocardium to some small extent, this is the exception. Malformed valves have less resistance than normal ones and are thus more prone to infection. The simple form is characterized by the development of minute verrucosities, often pediculated, upon the edge of the valve by preference, and more often upon the mitral than the aortic, and still less frequently in the valves of the right side. The epithelial cells of the affected region are damaged and a collection of blood plates and fibrin occurs here. In many instances some of the various micro-organisms mentioned are found in the vegetations. Proliferation of the subendothelial cells takes place later and a thickened area remains. In the acute diseases, for example rheumatism and chorea, the processes are more intense, and result in progressive changes leading to the thickening and deformity of the valve, which destroy its ability to occlude perfectly its orifice, or even lead to the more serious changes which cause obstruction. In the mildest cases of simple endocarditis the organization of the verrucose growths is so complete that the valve is restored to practically a normal condition.

In malignant endocarditis, the processes mentioned upon the valve margin develop a more serious condition. Micro-organisms are

always present, and ulcerative, and later necrotic lesions appear. The valves may become eroded, a diphtheritic-appearing deposit may be formed, or fungoid vegetations may become encrusted with lime salts. The damage to the valve may lead to perforation or rupture. If the mural endocardium become involved, similar accidents may occur even to the heart wall. In this type minute emboli are carried away in the blood stream, and septic infarcts may be found abundantly in the arterial tree. Inasmuch as the valves of the right side are more prone to become involved than in simple endocarditis, embolism may be found in the circulation of that side as well. The tendency of the malignant process to affect valves already damaged by chronic endocarditis has been mentioned. Many of the serious circulatory results of the severe forms of endocarditis are due rather to the associated acute degenerative type of myocarditis than to the valvular damage.

**Symptoms.**—These are often so trivial in simple endocarditis that the disease is not suspected. The fever of the disease with which the endocarditis is associated is not sufficiently modified to lead to suspicion of valvular infection. In rheumatic cases, it is common to have a feeling of distress in the precordium, with perhaps attacks of palpitation and a somewhat quickened pulse. Osler emphasizes the importance of fever in children, regarding it as by far the most important symptom. It frequently persists after the increased temperature of the rheumatic or other process has subsided, and is of great significance at this time. In adults the process may be entirely afebrile. In the recurring type of endocarditis seen in adults, pain may be a prominent symptom, and it may be of great severity.

**Physical Examination.**—Most important is the appearance of a murmur, most often at the mitral orifice. This must not be taken to be indicative of an endocarditis necessarily, since in many cases it is either purely hemic or the febrile disease has weakened the cardiac musculature and given rise to a murmur of relative leakage. Thus a systolic mitral murmur is very common in typhoid fever, in which endocarditis is relatively rare. A gradual increase of a very slight roughening, until in a few days a very definite murmur exists, which may tend to become rougher in character and is permanent,

and present in all positions of the body, is of evil omen. An unaccountable dilatation of the heart would render an endocarditis more probable, although occurring from associated myocarditis. An unusually forceful heart action is ground for suspicion that the endocardium is involved. A definite diastolic murmur at the aortic orifice may be taken with considerable certainty to indicate not only an endocarditis, but that the valve is already materially damaged. A musical murmur at the mitral region, in the same manner, is of more import than a soft blowing one, being generally associated with the presence of a vegetation. The presystolic murmur of mitral stenosis is almost never found until the post-febrile sclerosis of the valve has developed. I have seen hemiplegia from embolism as the first positive evidence of valvulitis. It should not be forgotten that the murmur may not become apparent until after the patient has been up for days or weeks, but this is not to be taken as evidence that the infective process has not involved the valves during the progress of the original disease.

**Malignant Endocarditis.**—This type is so closely associated with general septic involvement in many cases that the purely cardiac features are often obscured. It is common to describe it as being (a) septic, (b) typhoidal, or (c) cardiac in type.

(a) **SEPTIC ENDOCARDITIS.**—This is a feature of puerperal fever, pyemia, infected wounds, erysipelas, or other serious septic process, with the usual septic organisms of those diseases predominating. The pneumococcus and the gonococcus are not infrequently responsible. The picture is one of chills, sweats, high fever, delirium, coma, multiple embolisms of most varied type, with infection of the blood stream by the especial organisms involved. Petechiæ, retinal hemorrhages, optic neuritis, hematuria, pain from infarction of various organs, and even gangrene of a limb from embolism are features of this type. With so many septic manifestations the heart may be entirely overlooked, unless evidence of embolism attract attention. The course is rarely over a few weeks, and may be but a few days.

(b) **TYPHOIDAL FORM.**—This more commonly arises without an external focus of infection, as in pneumonia or gonorrheal infection

of the prostate or of the deep urethra. The symptomatology may so resemble that of typhoid that perhaps a majority of the cases are at first supposed to be that disease. A dry tongue, irregular fever, occasional chills, sweats, delirium, coma, diarrhea, parotitis and exhaustion are present, and in some cases no embolic signs appear. The heart may dilate and give rise to relative leakage in many severe febrile diseases, and this interpretation may be put upon the cardiac symptoms which finally turn out to be due to malignant endocarditis. Embolic manifestations are likely to constitute the deciding point. In the absence of blood cultures the early diagnosis may be impossible. In certain cases the cerebral manifestations are so marked that the disease is regarded as a meningitis, and in many cases a septic meningitis actually exists. The headache, retraction of the neck, cutaneous hyperesthesia, Kernig's sign, and even the results of spinal puncture may suggest a primary meningeal involvement. The fatal result commonly comes within a few weeks.

(c) THE CARDIAC GROUP.—In this type the malignant endocarditis occurs through an involvement, in an acute process, of an old valvular endocarditis. Several recurrences of a fairly severe form of endocarditis may have been noted in the history before the final malignant one. The cases may last many weeks, running a less severe course than the previously mentioned forms. The affection may be almost afebrile in character in rare instances. There is a chronic type of infectious endocarditis which may take a year to run its course, the symptoms being limited to fever and general failure for the first few months. Recurring chills may finally appear. Embolic features, which would commonly be decisive in diagnosis, are wanting until near the end, although the painful nodules which are occasionally seen are probably of embolic origin. A decided secondary anemia eventually develops.

Upon physical examination in cases of malignant endocarditis the heart may present signs in any degree, from trivial enlargement and a soft apical systolic murmur in the milder ones to enormous dilatation with great rapidity of action, and loud mitral and aortic murmurs in the more severe ones. I have noted the development of a diastolic murmur over the pulmonic valves in a slow type of purperal



sepsis, without signs of aortic regurgitation, and such involvement of the right side is probably not at all rare.

**Diagnosis.**—In the frankly septic type, the chief source of error lies in having the attention so sharply drawn to the general features of sepsis as to prevent the giving of due weight to the cardiac signs present. The severity of the process in the recognized cases is so much greater than in simple endocarditis that error is unlikely, although in cases in which a simple endocarditis finally becomes malignant there is a time when exact differentiation is impossible. Even its existence is at times impossible of immediate diagnosis. Thus a young negro entered my wards before the days of blood cultures and the Widal reaction, without any history of his illness. There were chills, high fever, delirium, many petechiæ, palpable spleen, dilatation of the heart to twice its normal area, and a loud systolic murmur at the apex, transmitted to the back. Death occurred before a blood count was obtained. The diagnosis of a most malignant type of endocarditis was made. The post mortem examination showed it to be the hemorrhagic form of typhoid fever. The heart valves were not involved (acute typhoidal myocarditis).

A close search should be made for petechiæ, for two or three red-dish-blue spots less than a millimeter in diameter may be sufficient to turn the diagnosis. In general the high leukocytosis and positive blood culture, with characteristic fever, and with no physical signs, excepting the cardiac ones and the embolic manifestations, render the diagnosis certain. Malaria is now easily excluded by the blood examination.

**Prognosis.**—It is practically always fatal excepting that occasionally one of the mild chronic types especially associated with recurring valvulitis may recover.

## B. SUB-ACUTE BACTERIAL ENDOCARDITIS

### (*Endocarditis Lenta*)

Libman and others in recent years have described a sub-acute bacterial endocarditis due, according to Libman, to the *Streptococcus viridans*. Rosenow claims to have cultivated the organism from the

pneumococcus. It has repeatedly been isolated from the blood stream. The vegetations tend to spread up on the left posterior wall of the auricle, and the chordæ tendinæ are often involved in the endocarditic process.

A striking feature of the affection is the involvement of the glomeruli of the kidneys as described by Locklein. The chronic nephritis often obscures the diagnosis of the cardiac condition. In many cases Libman found at post mortem examination the characteristic glomerular lesions associated with evidence of more or less complete healing of the endocardial process. The onset is insidious, with dyspnea, lassitude, fever, and vague joint pains. Osler found painful red swelling of the fingertips in certain cases.

A secondary anemia develops, and a fairly marked leucocytosis in most cases. A mitral murmur is nearly always present, the aortic valve being less constantly involved. Embolism is very frequently met with and a diffuse nephritis is almost constant. Sub-sternal tenderness is mentioned as a frequent finding, and many of the patients show a brown pigmentation, notably of the face. Splenic enlargement, petechiæ, and painful erythematous nodules are often noted.

The course in Major's cases varied between 3 and 24 months, all eventually proving fatal, as in Libman's series. Recovery, nevertheless, occasionally results, even after the demonstration in the blood of the causative organisms.

### C. CHRONIC ENDOCARDITIS

This process may occur either as a sequence of the acute form or as a sclerotic process from the beginning. In either event the essential features are a chronic sclerosing type of inflammation of the endocardium, especially of the valves, with such deformity of the leaflets as to prevent apposition, and leading to leakage, or to such adhesions and shrinking as to give rise to stenosis. The acute form of simple endocarditis has been considered. Its seriousness consists in the possibility of the development of a chronic form rather than in any damage done by the acute variety. The chronic form rarely

follows a malignant endocarditis because of the rapid course and usual fatal ending of the disease.

The second type of chronic endocarditis, sclerotic from the beginning, presents many of the features of arteriosclerosis. The degenerative changes are similar to those in the intima of the vessels, and sclerosis and even calcification occur as the process extends. The causes inducing arteriosclerosis are also operative in this form of valvular disease. High blood pressure, through the increased strain upon the valves, is a potent cause. Severe muscular strain, syphilis, chronic nephritis, gout, poisoning by lead and alcohol are all of importance. The aortic valves are especially susceptible to damage from syphilis and from the prolonged high tension of muscular strain. The left heart is affected in the vast majority of cases, and the auriculoventricular valves at least twice as frequently as the basic ones. The right side is chiefly involved in the fetus.

**Pathology.**—The delicate valve tissue loses its translucent appearance, becomes opaque and thickened, eventually sclerotic, and in extreme cases lime salts are deposited. The chordæ tendineæ become thickened, shortened, and may practically disappear. The sclerotic process may bind the edges of the valve segments together in such a way as to prevent their separation for the passage of the blood current, or roll up the hardened edge of the valve so as to prevent it from coming in sufficiently close apposition with its neighbor as to secure the orifice against leakage. Stenosis results in the former instance, regurgitation in the latter. In old sclerotic processes at the aortic orifice a calcareous outgrowth may so interfere with the passage of the blood stream as to produce the most violent vibration.

The symptomatology of chronic endocarditis will be considered under chronic valvular disease.

## 5. CHRONIC VALVULAR DISEASE

The effects of the involvement of the valves of the heart in the acute forms of endocarditis, which we have been considering, may be at once manifest, as in the more severe simple and malignant varieties. In most of the cases which recover from the earlier illness,

however, the symptoms which eventually are to develop from the progressive changes which have only been started in the valves do not show until months, and very often years, have elapsed. When finally the valves are so damaged that they fail to perform perfectly their functions, the patient comes under observation again, this time with the signs and symptoms of some of the different types of valvular disease.

In general the development of leakage or obstruction at an orifice as a consequence of valvulitis, does not immediately produce symptoms, because of nature's conservative management of the situation. In an arbitrary way we will assume that a healthy heart has sufficient reserve force so that it habitually uses but one-fourth of its normal power in a condition of bodily rest. The subject is able to do hard muscular labor involving two or three times the amount of work that the heart habitually does when the body is at rest, with still a fair amount of power in reserve. If a valve becomes so damaged as to interfere with the ordinary progress of the circulating stream of blood through the heart, dilatation of the cavity most affected occurs, and, with the increased demand for work in moving the larger quantity of blood contained, a response in the way of increased weight and strength of the muscular wall. In other words, hypertrophy of the heart has occurred. In certain cases the preliminary dilatation is not present. The heart is now able to do an amount of work above that of which a heart of normal size would be capable, although part of its extra force is wasted because of the valve lesion. Still its possessor can do the ordinary work of life with comfort, using a greater portion of his heart power habitually than does the healthy man, and having a less reserve than is normal. If content to limit his activities to such a degree that he never uses up all his reserve power, he is still able to follow the ordinary work of life without serious embarrassment.

So soon as the damage from the valvular lesion, which is unfortunately a progressive process as a rule, becomes so great that even with his hypertrophy he is unable to develop sufficient heart power for the ordinary calls of life, much less to keep any in reserve, his compensation is broken and he becomes an invalid to that extent.

Even in health one may use up all his reserve power by sudden severe exertion, as in an athletic contest, but the heart with rest quickly returns to normal provided the strain has not been of unusual severity, such as to produce an acute dilatation, the effects of which do not wholly clear up with rest. So a cardiac invalid may temporarily use up his reserve power and temporarily embarrass his compensation, but there comes a time when the "injunction is made permanent" and his compensation is "broken."

The incidence of valvular disease in 600 cases which I reported \* will be considered in the study of the separate affections.

### A. AORTIC INCOMPETENCY

**Etiology.**—Insufficiency of the aortic valves commonly results from one of two processes, either the sclerotic changes eventually developing as a sequence to the damage done to the leaflets by an acute endocarditis, or the arteriosclerotic type of degeneration already discussed. In addition to these two main classes there is a variety in which under strain an already damaged valve suddenly ruptures. A few cases result from the congenital malformation or fusion of the cusps, the deformed valves having an especial tendency to take on an endocarditic process. In conditions of extensive atheromatous degeneration of the aorta, in association with aneurism, and in extreme dilatation of the left ventricle, the aortic ring may stretch, a relative aortic incompetency resulting.

Aortic regurgitation following the chronic changes induced by an acute endocarditis is not uncommon in children and young adults. A much more common form is that associated with the arteriosclerotic changes of early and middle manhood. The causes especially operative here are those which tend, first, to lower the resistance of the tissues involved, and, secondly, to throw greater strain than normal upon the valves. The most prominent feature in the first class is beyond any doubt syphilis. Alcohol has been regarded as of importance, but the tendency now is to regard it as of perhaps

\* "A Clinical Study of Six Hundred Cases of Heart Disease," *Medical Record*, Nov. 13, 1909.

less significance in this lesion than it has been thought to be. The common cause of increased strain on the valve is sustained high arterial tension, such as is incident to the most laborious occupations. I was much impressed, when a house physician, with the number of stevedores and dock laborers who suffered from this form of valvular disease. With them periods of violent work, until the ship was loaded or unloaded, with alternating intervals of dissipation, gave an opportunity for all three of the factors mentioned to become operative. While relative leakage in case of a dilated atheromatous aortic arch is admitted, some clinicians dispute the occurrence of the type of dilatation, so frequent at the auriculoventricular openings, from the stretching of the ring. Yet many such cases have been reported upon good authority. I have several times noted a soft diastolic murmur of the utmost distinctness, with all the qualities of recognized aortic regurgitation, in men admitted with acute cardiac failure, which with rest in bed and digitalis cleared up. Others have repeatedly made similar observation. While undoubtedly rare as compared with an analogous lesion at the auriculoventricular orifices, there seems no doubt as to its occasional occurrence.

The reasons for the development of symptoms and signs in aortic incompetency are manifest. With the failure to close the orifice securely a small portion of the column of blood in the aorta leaks back into the ventricular cavity, which is kept under an abnormal tension to sustain the load. Reflexly the peripheral arterioles dilate at the time of systole in order to relieve the left ventricle of its abnormal load. The necessity of moving a greater volume of blood with each contraction leads to dilatation of the ventricle, and the requisite power for the work is provided by a marked hypertrophy. The heart lengthens and broadens as a result. It is not uncommon for it to double in weight, and instances of much greater enlargement are very frequent. With the increase in capacity of the left ventricle it is not unusual to have the mitral ring give way, with a resulting relative leakage. The frequency of a similar endocarditic process at this point must not be overlooked in considering the origin of an associated mitral murmur. As a result of the relative or organic leakage through the mitral orifice, the left auricle suffers dilatation,

secondary hypertrophy occurs, and in the severe types the right side of the heart is eventually enlarged, first by dilatation, then by hypertrophy. A limit is set to the hypertrophy of the heart because of the impossibility of nourishing a continually growing muscular organ through the vessels designed for a normal use. The failure of the usual filling behind the aortic cusps, where the entrances to the coronary arteries are placed, is a factor in this form of valvular failure. A more serious one lies in the tendency for the atheromatous process to reach and obstruct these arteries, with inevitable danger to the nutrition of the muscle walls, as discussed under myocarditis. The increased strain put upon the aorta and the smaller arteries by the tremendously sudden expansion to which they are subjected tends toward the development of arteriosclerosis in their walls. The aorta is enormously dilated in some cases and may cause the diagnosis of aneurism to be made.

**Symptoms.**—During the stage of good compensation the patients often follow laborious occupations without knowledge of their disease. Compensation may be as perfect in this condition as in any valvular defect. With the first signs of failure the patient may complain of the throbbing in his chest or head, of giddiness or of faintness on suddenly rising, and of distress about the heart, especially on exertion. Attacks of palpitation are not uncommon. Dyspnea is rarely present until the heart failure has advanced far enough to permit of passive pulmonary congestion. The patient, in the severe cases, has an ashen pallor, which is fairly characteristic, especially when contrasted with the congested face of mitral disease.

More than in any other valvular disease precordial pain is noted, and it may be severe or even agonizing in character. Irritability, sleeplessness, and melancholia are not rare.

If the valvular process be inflammatory in type, as is often the case, irregular fever may be present over long periods.

After a definite break in compensation the symptoms are modified by the addition of certain features which pertain to the affection of the other valves and cavities. Hemoptysis, which is rare as a symptom of aortic regurgitation during the stage of good compensation, may occur as the result of the passive congestion of the

lung following mitral involvement, and cough and expectoration are often present. Edema and finally general anasarca eventually appear in a few cases, after the mitral valve and the right heart are over-dilated. Cyanosis, dyspnea, and even orthopnea arise chiefly from the same source. Embolism in the general arterial distribution may occur. Finally, aortic regurgitation has the unenviable distinction amongst valvular diseases of being the one most frequently ending in sudden death. This may occur from embolism, sudden dilatation, or an anginal attack.

**Physical Examination.**—**INSPECTION.**—The first feature to attract attention in many cases is the throbbing which results from the violent filling of the arterial tree by the powerful heart. The head is seen to “bob,” the foot oscillates widely if the knee be crossed over the other one, and the arteries of the neck and even of the head and face may be seen to pulsate strongly. In the stripped patient the throbbing of the brachial and femoral arteries is often seen to be very marked. Upon grasping the arm the whole mass may be felt upon careful observation to pulsate within the hand.

Upon examining the precordia one sees the left side of the chest bulged in the younger subjects and, as a rule, a wide and forcible area of cardiac pulsation. The apex-beat may be visible in the sixth or seventh space, anywhere between the mammillary line and that of the anterior axillary border. In this, the largest type of heart which is recognized clinically, the “*cor bovinum*” of aortic regurgitation, the area may be even greater. The impulse is found to be strong if the compensation be still good, and the contraction of the heart gives a peculiar and powerful heaving sensation to the hand. After secondary dilatation has occurred the beat is weaker and the usual wavy character is noted in the impulse.

Upon percussion the enormous area of the cardiac dulness is appreciated. It extends farther to the left in extreme cases, than in any other condition. With the stethoscope one may hear one of the most characteristic of all heart murmurs, the long blowing diastolic murmur beginning at the region of the aortic cartilage and extending downward either toward the apex or along the right side of the sternum. Its exact line of propagation is probably in part



dependent upon the situation of greatest defect in the aortic cusps. When the leakage is slight the murmur may be heard with the ear or the phonendoscope when inaudible with the stethoscope, the former means gathering up sound waves over a much wider territory. Upon the extent of the damage to the aortic valves depends the presence or absence of the aortic second sound. If all three cusps are sclerotic so that no flapping of the valve is possible, only the regurgitant murmur is heard, while if a single valve retains the power of closing with the proper "snap," an accentuated aortic sound may be present.

Because of the roughening of the aortic orifice a systolic murmur is very commonly heard there, often transmitted into the neck, but stenosis is not to be lightly assumed. It is more frequent in the cases secondary to a rheumatic endocarditis than in the atheromatous type. A diastolic thrill traceable downward along the sternum is not infrequent in this disease (twice in my series), and occasionally a double thrill indicates not only leakage but stenosis. The apical first sound is clear unless mitral trouble is present. An exception to this rule exists in the case of the so-called Flint's murmur. Here a presystolic murmur much resembling that of mitral stenosis is present. It is best explained as being due to what might be called a relative narrowing of the mitral orifice, caused by the floating out into the incoming auricular stream of the large anterior mitral flap by the regurgitating current from the aortic orifice. Thrill even may be present, but the sharp first sound of true stenosis is rarely to be found. I have several times made the diagnosis of coincident mitral stenosis in cases of aortic regurgitation and proved it at post-mortem. The Corrigan pulse is, I believe, less marked in these cases; the signs of mitral stenosis, including the pulmonic accentuation, are well marked, and hemoptysis or dyspnea may have been noted in the history before any signs of failure from aortic lesion had occurred. The arteries are commonly somewhat less sclerotic.

The striking feature in examining the pulse is its "water hammer" quality. This is best appreciated by having the patient elevate the arm above the head and then grasping his forearm above its middle, with the palm of the hand over the radial and ulnar arteries.

The characteristic "slap" of the artery against the hand in these cases practically suffices for the diagnosis. Upon examining the retina with the ophthalmoscope the pulsation of the arteries may be noted. The ophthalmologist occasionally makes the diagnosis and sends the patient to the physician. By pressing one corner of the fingernail in such a way as to obtain varying degrees of anemia of the nail-bed the capillary pulsation may commonly be exquisitely demonstrated. It may be visible in the lips, along the hyperemic edges of the line resulting from drawing the pencil across the forehead, or, even without such an expedient, in the normal skin. The pulsation in extreme cases goes through the capillaries into the peripheral veins. The sphygmographic tracing is exceedingly characteristic. It shows the abrupt line of ascent and the sudden fall unless the latter is modified by the arteriosclerosis. The pulse wave at the wrist is delayed in accordance with the degree of regurgitation. The systolic pressure, owing to the hypertrophy of the left ventricle, is high, but the diastolic is decreased. The double murmur over the femoral artery, upon slight compression (Duroziez's sign) and the "pistol shot" heard as the systole begins, are of interest rather than of importance. The marked pulsation of the aorta, lifting the whole abdomen, should be mentioned. The diagnosis of aneurism is occasionally made.

**Diagnosis.**—This is the easiest amongst those of the valvular diseases. The great enlargement of the heart in well-developed cases, the character of the murmur, which is perhaps the most reliable in diagnosis of any found in the heart, the visible pulsation in the arteries and the Corrigan pulse should suffice. The capillary pulsation may occur also in neurasthenia, in exophthalmic goiter, and other conditions of marked peripheral arterial relaxation, but care in the examination prevents error. The dynamic expansion of the aortic arch in young persons with regurgitation suggests aneurism. The examination with the fluoroscope may be of more service than the X-ray plate in these cases. The infrequent regurgitant murmur at the pulmonary orifice should be mentioned, although it could not be confused with that of well-marked aortic incompetency. The existence of pulmonary leakage only as a congenital trouble, after

septic endocarditis, or in connection with very advanced mitral stenosis or fibroid phthisis, as a rule, should be borne in mind. The latter type must be relative in character. This diagnosis is to be made with caution, however, and any signs pointing toward the circulation of the left side of the heart should be given especial consideration, in such differentiation.

In case a diseased aortic cusp ruptures suddenly, the diagnosis can scarcely be mistaken. Several times I have found a patient with endocarditis worse in the morning, with all the signs of a violent aortic regurgitation which had developed over night. The dilatation of the heart is extreme and physical examination is decisive.

There were 49 instances of aortic incompetency in my series.

**Prognosis.**—Recovery never occurs in case of organic leakage, so far as we know. The patients may go for years without a sign of failure, but after an interruption of compensation occurs the outlook is not good. The arteriosclerotic patients are likely to suffer from damage to the aorta or coronary arteries, or from angina pectoris. Recurrent endocarditis is a serious menace in some cases. As in the case of the mitral valve, a moderate narrowing combined with regurgitation makes the outlook more favorable. In rupture of the valve, death commonly occurs within a few weeks, since little or no compensation on the part of the heart is possible under the suddenly developed conditions. The prognosis, in case the Wassermann reaction is positive, is somewhat more favorable, since specific treatment may be of some avail.

## B. AORTIC STENOSIS

Roughening from sclerotic changes is common at the aortic orifice, but actual narrowing is comparatively rare. In many cases a certain amount of leakage is also present. Typically the disease is found in arteriosclerotic men beyond middle age. The narrowing is commonly the result of a slow sclerotic process with calcification superadded. The valve segments become adherent to each other so that they cannot be pushed out of the way of the emergent blood stream, obstruction to its passage resulting. The condition may also

follow acute rheumatic endocarditis, the sclerotic process progressing with deposit of lime salts until the whole aortic ring is a solid mass excepting for the narrowed orifice. Leakage necessarily occurs in this type. Because of the increased work necessary to force sufficient blood through the narrowed orifice the muscular wall of the left ventricle hypertrophies. Since there is no dilatation the hypertrophy is of the "concentric" type, existing around a ventricular cavity of normal size. The heart may be much increased in size. If coincident aortic leakage exists the character of the ventricular change is modified, approaching that of aortic regurgitation. A prolongation of the period of systole is noted, since the obstruction makes the passage of the blood slower, notwithstanding the hypertrophy of the ventricle. The blood pressure is not noticeably affected. When finally the obstruction becomes so great that the ventricle cannot completely empty itself, dilatation begins, with relative leakage at the mitral orifice, and the whole chain of troubles that follows broken compensation in the left heart. The arteries, notwithstanding the average greater age of the patients, are less damaged than in aortic regurgitation, having been spared the strain of the sudden expansion of that affection.

**Symptoms.**—These are less characteristic than in any of the other forms of valvular disease. Aside from transient dizziness, especially on suddenly rising, headache from anemia of the brain, slight dyspnea on exertion, lessened cardiac reserve, with distress upon drawing upon this reserve, there are no symptoms in the uncomplicated cases. They may go on for years in comfort. One patient in the Denver City hospital, with pronounced murmur and thrill, and in whom I found post-mortem an extreme degree of narrowing of the orifice, was shown annually at clinics by Dr. Sewall and myself for many years, yet was able to work about the hospital. His death, at nearly 70 years of age, was incurred through exposure when he was temporarily out of the hospital and became intoxicated. After compensation is lost the usual train of symptoms in cardiac cases develops. Embolism is an occasional feature.

**Physical Examination.**—Pallor may be noted at the time of the attacks of vertigo. If not concealed by the emphysema, common in

old men, the heart area is found to be moderately increased in typical cases, and the apex-beat is found slightly lower and more to the left than normal. Yet the changes in these directions are surprisingly small in comparison with the extent of the damage to the valve, and in many cases are scarcely noticeable. The apex-beat may be somewhat forcible, but may be even imperceptible. The most important feature for the diagnosis, although not so often present as the murmur, is the systolic thrill felt over the aortic area, and transmitted upward (four times in my series). As in the patient mentioned, it may be extremely rough, and in such cases a systolic murmur of incomparable harshness accompanies it. This is also transmitted upward into the vessels of the neck. It may be heard at a distance and is often of musical character. In the absence of well-defined thrill and of typical symptoms, the systolic murmur is more safely attributable to roughening of the orifice or the ascending portion of the arch. Owing to the sclerosis of the aortic valves the second sound is not infrequently wanting. If coincident regurgitation be present the diastolic murmur is added and the harsh character of the stenotic murmur is often modified. The pulse is commonly small, of good tension, and often slow. As regurgitation develops or the heart muscle fails, signs of leakage at the mitral orifice appear with the usual general circulatory failure.

**Course.**—As in other conditions involving extreme degrees of sclerosis about the aorta, angina pectoris, coronary arterial trouble, myocarditis, and the various features which have been discussed under that heading are not infrequent.

**Diagnosis.**—This has been sufficiently dwelt upon. The reader should remember that the condition is a rare one; that at least 20 to 50 cases of systolic murmur are to be found for every case of definite stenosis; and that thrill and murmur of very harsh character are essential to the diagnosis unless the symptoms be plain enough to justify it in the absence of thrill. The diagnosis was made clinically but eight times in the 600 cases of heart disease quoted.

**Prognosis.**—This may be said to be dependent rather upon the general age and arterial condition and the possibility of the development of angina or myocarditis, or the addition of mitral leakage

with its serious results, than upon the valvular lesion proper. In the young the type following acute endocarditis is decidedly serious.

### C. MITRAL INCOMPETENCY

**Etiology.**—The usual cause lies in the sclerotic changes which develop in the leaflets as the result of a chronic endocarditis which has supervened upon the acute form, most often of rheumatic origin, but also originating in the sclerosis of advancing years. The thickening and crumpling of the valves results from the shrinking of the newly formed connective tissue within the valve, a product of the endocarditis. The shortening and even disappearance of the chordæ tendinæ has been mentioned. Lime salts may be deposited. Stenosis of the orifice is not infrequent.

In a greater number of cases the disease arises from a totally different cause. The mitral valve, unlike the basic valves, is supported by a ring of muscular tissue. In any condition of exhaustion, be it from severe exertion, acute disease, or chronic debilitating conditions, the muscular tissue, as the ventricle dilates, weakens and yields, and the valves can no longer close the orifice, since it has dilated. After restoration to health and even after physical rest, the normal conditions may be restored. Weakening of the papillary muscle is a more important feature in the production of the insufficiency.

**Effects of the Lesions.**—If the leakage be of a type which persists, whatever the original cause, the first trouble is felt by the left auricle. This receives back a portion of the blood it has thrown into the left ventricle, but meanwhile has drawn a full charge from the pulmonary veins. It dilates to make room for the added quantity of blood and must hypertrophy to be able to handle the increased amount regularly. The left ventricle, meanwhile, in order to send forward the normal amount, must receive more and handle more than normally, albeit it wastes its efforts so far as the part thrown back into the auricle is concerned. With the changed auricular conditions, the entrance of blood from the lungs is rendered more difficult, and the intrapulmonary pressure is raised. This necessitates an

increased power on the part of the right ventricle, and hypertrophy with a certain amount of dilatation occurs. If the tricuspid valves give way, and they always do sooner or later, similar changes take place in the right auricle, and general venous stasis is the final result. In the lungs dilatation and atheromatous changes occur as the result of increased pressure, and the condition spoken of as brown induration is the eventual development. In similar manner, through the systemic veins, the stasis which results after the right ventricle and auricle have dilated gives rise to the cyanotic induration of the viscera.

In general the relative incompetency is much less perfectly compensated and in the severe types may go on to a more rapid course. The cause of the leakage, whether organic or relative, cannot as a rule be decided if the case be seen without history after the compensation be broken.

**Symptoms.**—In the ordinary cases the development of the lesion has been so gradual that compensation has been established step by step with its increase, and there may be no symptoms. One of the great professional bicycle racers of 15 years ago had a loud mitral regurgitant murmur and marked hypertrophy of the heart, and yet was able to ride short races successfully for years. In general there is a lack of the full cardiac reserve so that exertion gives rise to moderate dyspnea, which is the earliest symptom in most cases. This stage of practically perfect compensation may last for years. Finally from increase of the degree of leakage, injudicious exertion, acute disease or insufficient nutrition, the heart fails to do its work easily, and more decided dyspnea is felt even after quite moderate exertion. The venous stasis becomes more pronounced and slight recurrent hemoptysis is not infrequent. Because of the poor pulmonary circulation an ordinary bronchitis is prolonged, and cough and expectoration last perhaps most of the winter. The veins of the face and ears and of the feet and hands become slightly dilated, and a slightly cyanotic color results, and even quite definite cyanosis in cold weather. The hands and feet do not keep warm easily, and clubbing of the nails develops. The urine becomes somewhat scanty and the general endurance is lessened. Palpitation is an occasional

symptom, and from the venous congestion, hemorrhoids and, in women, menorrhagia may be noted.

Eventually a definite break in compensation occurs, and the symptoms become more marked. The reserve power of the heart fails and dyspnea follows the most moderate exertion. More marked cyanosis develops, the feet become slightly edematous, and upon exertion markedly so. The engorged lungs may become dropsical, and in the extreme cases hydrothorax, most often upon the right side, and ascites, follow the dropsy of the extremities. Because of the chronic passive congestion of the liver a slight jaundiced color becomes apparent.

The sleep is poor, largely on account of the dyspnea. Upon falling asleep the patient suddenly starts up frightened, and is afraid to go to sleep again for fear of losing his breath. The urine contains albumin and a few hyaline and granular casts, due to the passive renal congestion.

Although the patient may, under rest and treatment, recover sufficiently to be up and around for some months, even on several different occasions, permanent disability eventually occurs.

**Physical Examination.**—**INSPECTION.**—The cyanotic countenance and clubbed fingers attract attention. Children are commonly several years behind their proper growth, as judged by their age, in the cases which have started early in life. The left side of the chest is more prominent if the disease has begun in childhood. The apex-beat may commonly be seen to the left of its normal place, and possibly in the sixth interspace. A wavy impulse is commonly noted over the precordia. Because of the hypertrophy in the right ventricle epigastric pulsation is frequently noted. Dropsy is apparent. The liver is visibly enlarged. The cervical veins are overfull, and may pulsate if the tricuspid valve has given way. Upon palpation a systolic thrill is occasionally found at the apex. It was present in 11 of my 348 reported cases of mitral regurgitation. The liver may be felt below the ribs in most cases. Percussion shows a marked increase in the transverse diameter of the heart, and upward toward the second space, and less in other directions. The region of the base of the lungs may be flat from double hydrothorax.



With the stethoscope one hears the typical soft, blowing apical systolic murmur in most cases. It often replaces the first sound and is commonly loudest at or near the point of the apex-beat. It lasts through systole, and is transmitted to the left and often into the back. The murmur is more often musical than any other, but this is of no especial significance. It may vary greatly from the normal in many ways. Not infrequently it is transmitted upward toward the left shoulder. It is perhaps loudest in the recumbent posture in most instances. The beginning of the murmur may be delayed an instant after the first sound, the late systolic murmur. A rumbling presystolic murmur may precede it, indicating associated stenosis. The systolic murmur may be fainter at times owing to cardiac exhaustion. The pulmonary second sound is commonly accentuated and may be heard well transmitted to the apex. If there be pulsation of the veins of the neck or of the liver, the systolic murmur of tricuspid regurgitation may be heard near the lower end of the sternum. Clinically the most common association of irregular pulse is with mitral regurgitation in the stage of broken compensation. It is commonly due to the nodal rhythm. The blood pressure often falls below normal at this time. The loudness of the murmur has no relation to the degree of leakage, unless we may say that with a dilating heart it decreases with the increase in the degree of insufficiency.

**Diagnosis.**—This is ordinarily attended with little difficulty in the usual cases, for we find a well marked systolic murmur, transmitted to the left, and with an accompanying accentuation of the pulmonic second sound, and the other features described. It may be impossible to state whether the leakage is organic or relative, but the history and the accompanying features generally suffice for this. If the murmur completely disappears under rest and digitalis we may consider it of relative character. Accidental murmurs are very common at this orifice. The absence of symptoms, and of enlargement and accentuation of the pulmonary second sound are sufficient to exclude an organic leakage.

Mitral regurgitation was diagnosed clinically in 348 of the 600 cases quoted.

**Prognosis.**—This may be extremely variable. In general in the organic cases it is favorable for a considerable length of life if the patient only exercise proper care. A physician of my acquaintance suffered from mitral leakage following acute rheumatism for more than 45 years, and finally died of cerebral hemorrhage. The outlook in relative leakage depends entirely upon the condition which has given rise to it.

Embolism is less common in mitral regurgitation than in stenosis, but I have reported a death from blocking of the aorta at its bifurcation, presumably by a ball thrombus from the left auricular appendix.

#### D. MITRAL STENOSIS

**Etiology.**—The most frequent cause is the sclerotic contracting process initiated by the acute endocarditic process of rheumatic fever or of chorea. In a considerable number of cases no cause is recognized, and it is attributed to a similar chronic sclerosis of unknown origin. It is more frequent in women, since rheumatic fever and chorea are more often found in the female sex. Anemia, whooping-cough, scarlet fever, and other causes have been suggested. From analogy it seems probable that some infective process is the origin of the trouble even in those cases in which it is not recognized. One difficulty in tracing the cause is found in the fact that mitral obstruction does not originate as a manifestation of the acute endocarditis, but only as the result of the slow sclerosis induced, while in the case of mitral insufficiency the acute process may lead to leakage even in the first few days of the trouble, and thus lead to the association of the valvular defect with the infectious disease present.

The valve is puckered and contracted until, in the typical cases, a mere "button hole mitral" is left, oftentimes at the bottom of a funnel, as looked at from the auricular side. Lime salts are often deposited so that no valve action is possible. The chordæ tendineæ are generally damaged. Regurgitation is present in this type, and to some extent in probably nearly all cases of stenosis. The extension of the sclerotic process to the muscular wall is not infrequent, and the incidental damage to the auriculo-ventricular bundle is the

cause of the heart's irregularity, to be mentioned later. The heart is only moderately enlarged, rarely to double its weight. The increased work thrown upon the left auricle and the right cavities as compared with the smaller burden of the left ventricle leads to a corresponding discrepancy in the size of the respective cavities and the thickness of their walls. Any changes in the left ventricle are due to the often associated mitral incompetency. Ball thrombi are not infrequently found in the dilated auricle.

**Effects of the Lesion.**—Because of the obstruction to emptying itself into the left ventricle the wall of the left auricle must hypertrophy. It is in fact often two or three times as thick as normal. The retention of an abnormal amount of blood causes dilatation, and the backing up into the pulmonary veins with the throwing of the burden on to the right side of the heart is the same process described under mitral incompetency. In favorable cases the restoration of the balance in the circulation through the left auricular and right ventricular hypertrophy is surprisingly perfect even for a period of many years.

**Symptoms.**—Because of the facts mentioned in the last sentence one need not be surprised to learn that many cases of mitral stenosis are practically without symptoms for a long period. Careful inquiry generally reveals that shortness of breath upon severe exertion has existed for perhaps a number of years. I have seen many cases of this affection sent to Colorado for supposed tuberculosis, in whom no especial cardiac symptoms existed, excepting the dyspnea and several slight hemoptyses, the latter being the cause of the alarm over the supposed pulmonary condition.

Girls with mitral stenosis do not develop properly, and puberty is often delayed. Slight cyanosis, cough, especially in winter, and frequently moderate anemia are present in the more severe cases. A history of recurring attacks of endocarditis may be obtained. When the break in compensation comes, all the symptoms are accentuated, and hemoptysis is likely to be more frequent and severe. The edema of the feet, hydrothorax, or general anasarca which appear are associated with the venous stasis dependent upon the involvement of the right heart, rather than directly with the original

valvular lesion. I have seen two cases of mitral stenosis complicated with recurring attacks of acute edema of the lungs, with expectoration of much pinkish, serous fluid. One of them practically drowned in his second attack from inability to keep his lungs clear by expectoration. Disproportionate activity of the hypertrophied right ventricle is the supposed cause. Embolism is probably more common in this form of valvular disease than in any other. Hemiplegia and infarction of the various organs may occur.

**Physical Examination.**—The precordial area is often less prominent than in connection with the regurgitant lesions. Owing to the predominating hypertrophy of the right ventricle the impulse is likely to be within the mid-clavicular line or even in the epigastric region, but it may be well outside the normal position. The area is less increased than in regurgitation. A wavy impulse is not uncommon in the region below the pulmonic area. With failing compensation an increase in the cardiac area and dilatation of the cervical veins may be noted.

Palpation reveals in many cases a presystolic thrill, especially during expiration, generally felt just within the point of the apex-beat. It was noted 22 times in the 79 cases of stenosis quoted. The thrill often disappears during incompensation. The shock of closure of the tricuspid valve is believed to explain the abruptness of the first sound, and the force of the apex-beat as felt with the hand. The rough thrill and the shock mentioned ordinarily suffice for the diagnosis even in the absence of all other signs and symptoms. Palpation over the area of pulsation mentioned, at the third or fourth left costal space, reveals the pulsation of the conus arteriosus of the right ventricle.

In percussion, because of the hypertrophy and dilatation of the right ventricle, the area of precordial dulness is found to extend two or three finger breadths beyond the right edge of the sternum, and in well-marked cases well beyond the left mammillary line. The dulness is high to the left of the sternum, and the large left auricle is well shown upon the X-ray plate.

Auscultation reveals over a small area, generally just within the point of apex-beat, the characteristic murmur of stenosis. It

corresponds in time with the auricular systole, and has a blubbery, rumbling quality that suffices for its identification, after it is once thoroughly appreciated. No other lesion causes such a murmur excepting in the rare cases of tricuspid stenosis. The occurrence of the softer diastolic murmur, often heard in the first half of the diastole, is attributed by Mackenzie to the flowing of the accumulated blood through the damaged valve before the actual occurrence of the auricular systole. It is very different in quality from the rough crescendo murmur of that period. This terminates with the occurrence of the sharp first sound of the heart, which may even be audible several feet from the patient. Because of the accompanying hypertrophy of the right ventricle and high pulmonic tension, a sharp accentuation of the pulmonic second sound is present. It is generally louder than in mitral insufficiency. Reduplication of this sound is regarded as highly significant of stenosis by many clinicians. In extreme cases the murmur of relative insufficiency at this valve is found after exertion, a soft diastolic murmur transmitted downwards from the pulmonary area, and *without any of the signs of aortic insufficiency*. There can be no question of the comparatively frequent detection of this murmur if it be sought for repeatedly in every case of stenosis. Its general transient appearance should be noted. The murmur of mitral leakage is often associated with that of stenosis, especially in failing compensation. The frequency with which the stenotic thrill and murmur disappear during incompensation, to be replaced by an apparently pure regurgitation, and to reappear after rest and digitalis, can scarcely be appreciated until one has personally noted it. The systolic murmur of tricuspid regurgitation is often found at the tip of the sternum. In a case of mitral stenosis recently seen with Dr. Henry Sewell he diagnosed an associated tricuspid stenosis with a separate presystolic murmur and thrill, and it was proven at post-mortem. These cases are very rare.

After incompensation is established the heart becomes irregular, often extremely so. This is due to the damage to the fibers of the auriculoventricular bundle, distributed over the walls of the auricles, inordinately stretched with the great dilatation, and is accompanied

by auricular fibrillation, but in certain cases, perhaps, to damage nearer the auriculoventricular node. Nodal rhythm is thus established. The action of the left auricle being no longer efficient under the conditions of nodal rhythm, the presystolic murmur disappears for a time.

**Diagnosis.**—This is easily established if the case be observed when the pathognomonic presystolic thrill and exaggerated first sound are present. If compensation has failed, the case is likely to be mistaken for one of ordinary mitral regurgitation. The pulsation in the region of the pulmonary artery, already mentioned, may simulate aneurism, the more so since left recurrent paralysis is an occasional feature. It is due to direct pressure of the enormously dilated left auricle, or transmitted pressure pinching the nerve between the pulmonary artery and the aorta, and establishing a pressure neuritis. The presystolic murmur of aortic regurgitation (Flint murmur) should be credited to that lesion, except in case the clearest evidence in favor of mitral stenosis is present. Indefinite presystolic murmurs have been mentioned in cases of pericardial adhesions, but they are said to lack the sharpness and other features of the stenotic murmur.

There were 79 instances of mitral stenosis in my series.

**Prognosis.**—The cases may live nearly an ordinary lifetime if compensation remains fairly perfect. The danger of embolism has been mentioned. Pneumonia is peculiarly fatal to these subjects because of the embarrassed heart action. I have seen but one recovery under these conditions, and I have several times known the fatal result to occur in from 36 to 72 hours. After compensation is broken, the prognosis is comparable to the similar condition in mitral regurgitation.

## E. DISEASES OF THE PULMONARY VALVES

**Stenosis.**—This occurs chiefly as a congenital affection which will be discussed more fully in the appropriate section. As an acquired condition it is rare, and it possibly occurs upon a congenital basis in some of these cases. It may be found as an ordinary endo-

carditic process, or as a sclerotic one. In the latter case an acute infection may be superadded. The diagnosis must rest upon the demonstration of a systolic murmur over the pulmonary region, and not transmitted into the vessels of the neck. In certain cases it may be heard in the back at a point opposite the pulmonary artery in front. A vigorous systolic thrill adds greatly to the presumption of correct diagnosis. Hypertrophy of the right heart may be found. The sound of closure of the pulmonary valves is commonly wanting. Non-stenotic murmurs of this region are extremely common, and may be due to unusual direction of transmission of the murmur of mitral regurgitation, to anemia or to fever. The cardiorespiratory murmur is frequently found here.

No especial importance is to be attached to a systolic murmur here without decided symptoms, or the signs of an actual stenosis. If thrill be present, the murmur deserves more consideration. Dyspnea may be expected in case of obstruction, and the signs of general circulatory failure may appear if compensation become broken. Clubbed fingers are often noted. The poorly nourished lung is liable to become the seat of tuberculous infection.

There were five cases diagnosed as pulmonary stenosis in my series.

#### F. PULMONARY INCOMPETENCY

This is most frequently found as a congenital affection, but may be acquired, especially in malignant endocarditis. A diastolic murmur occasionally associated with thrill develops at the pulmonary area and is transmitted toward the apex of the heart. It is said to be rougher and more superficial than the corresponding murmur at the aortic orifice. The signs of aortic regurgitation are absent. Any symptoms which arise are traceable to the dilatation of the right ventricle, and are similar to those noted under pulmonary stenosis.

Relative insufficiency has been mentioned in connection with mitral stenosis. A soft, inconstant diastolic murmur transmitted downward from the pulmonary area presumably signifies this condition if any cause of markedly increased pulmonary tension be pres-

ent. Gibson's experimental work upon animals, showing the great ease with which leakage occurs with increase in the pressure in the pulmonary artery, forms a sufficient basis for the statement that the regurgitation at this orifice is probably very much more frequent than the opinions of many clinicians would lead one to suppose. I have certainly watched the development of a diastolic murmur at this region several times in severe cases of mitral stenosis, and of fibrosis of the lung, and noted the disappearance and reappearance under varying conditions. In one such case which I watched for years the pulmonary valves were entirely normal at post-mortem.

There were seven cases diagnosed as pulmonic regurgitation in my series.

#### G. TRICUSPID VALVULAR DISEASE

Stenosis is rare, occurs most frequently in women, resulting from acute endocarditis as in the mitral and aortic valves, and is associated with mitral stenosis in more than half of the cases.

The symptoms arise from the inability of the right auricle to empty itself, even though hypertrophied. The auricular pulse in the cervical veins persists so long as the auricle works under favorable conditions, but disappears with extreme dilatation. Further symptoms relate to the backing up of the systemic venous circulation, cyanosis and dyspnea being most marked and often of extreme severity. There may be polycythemia, even eight to nine million red cells. Examination may show a presystolic murmur and thrill, separate from the same signs farther to the left and due to the associated mitral stenosis. Presystolic (or auricular) pulsation in the veins of the neck and the liver may be detected. It is important to bear in mind the fact that systolic venous pulsation signifies that the tricuspid valve has given way from dilatation, and the pulsation is therefore ventricular in origin, while the presystolic pulsation occurs only when the valve protects the auricle from a ventricular regurgitating wave, the pulsation being then synchronous with the auricular contraction. Since stenosis acts to prevent serious regurgitation the latter type of pulsation is found with it. The murmur



has the same character as the left-sided stenotic murmur, and the same systolic shock at the time of valve closure. The heart dulness upon the right is increased. The valves in the jugular veins have been heard to close with a "snap" audible with the stethoscope (Mackenzie). The diagnosis is rarely made during life (*see Mitral Stenosis*).

There was no diagnosis made of this affection in my series.

#### H. INSUFFICIENCY

Regurgitation at the tricuspid orifice may be organic in character, but is so common as a relative condition that its organic nature could scarcely be diagnosed during life. The valve becomes insufficient upon any material increase of the heart's action, and obviously was intended to become so. The mere statement that in experimental animals it is impossible to raise the blood pressure in the right heart because of the constantly induced tricuspid regurgitation is extremely significant.

Relative leakage occurs in many chronic diseases tending to increase the work of the right heart, notably mitral stenosis, emphysema, fibrosis of lung, chronic bronchitis, etc. The failure of compensation in the right ventricle is commonly due to this regurgitation. The patient complains of breathlessness, often extreme. Orthopnea and oppression in the chest are common. Dropsy and passive congestion of the abdominal organs supervene. The liver may be painful because of the stretching of its capsule, due to the great passive congestion. Intense cyanosis is present in extreme cases. With this are found marked dilatation of the veins of the neck and arms and the bulbus jugularis may be very prominent. The venous pulsation is systolic in type, since the auricle is over-distended and incompetent. Systolic pulsation of the liver may be extremely marked. A systolic murmur, generally soft and blowing, is heard at the lower end of the sternum, occasionally transmitted to the right, and differing in timbre from that of the mitral valve, if mitral insufficiency coexists. Roughness of the murmur and the addition of a thrill would suggest an organic lesion. The area of the

heart is increased in the region of the right ventricle, and the hypertrophy of that cavity may give rise to epigastric pulsation. Stern observes "that lowering the head with the body recumbent causes distension and pulsation of the jugulars, but if the head is lowered enough to stretch these veins pulsation and engorgement recede. This latter posture brings out the characteristic regurgitant murmur in the tricuspid area and also the venous hepatic pulse." Regurgitation at this orifice is of little importance clinically, as it is generally entirely overshadowed by the serious lesions with which it is associated.

Tricuspid regurgitation was diagnosed 14 times in my series, but relative leakage was doubtless very much more frequently present at some period.

### I. COMBINED VALVULAR LESIONS

In nearly half of the cases of valvular disease, at some time in their course, the evidence of lesions of more than one valve may be detected. In my series the most frequent combinations were as follows: Mitral stenosis and regurgitation existed together 44 times; mitral and aortic regurgitation, 31 times; mitral and tricuspid regurgitation, 8 times; other combinations were very much less frequent.

In discussing the valvular diseases, we have repeatedly spoken of the changes in the heart which lead to the involvement, organic or relative, of another valve. If one or two organic lesions be made out, the addition of further murmurs is likely to be due to the disorder into which the circulation is thrown. Yet a definite and correct diagnosis of double organic lesions at both aortic and mitral orifices is not rare.

**Prognosis.**—The outlook in chronic valvular disease turns not upon the nature of the murmurs, but upon the condition of the heart muscle, the efficiency of compensation, and the likelihood of recurrence of endocarditis. Osler's statement that, if the heart be regular and the apex-beat be normal in location, the auscultatory phenomena may be practically disregarded disposes of doubt in many cases. More and more attention is being paid to the condition of the

heart muscle and less to the murmurs. As to the valves involved, affections of the mitral are much the most favorable, and regurgitative lesions are more favorable than stenotic ones. Mitral lesions occur earlier in life, and the patients are thus less subjected to the results of the degenerating influences of arteriosclerosis; they occur more commonly in women, who are less exposed to over-exertion and the evil effects of alcohol, tobacco, and syphilis. These advantages more than counterbalance the disturbances of pregnancy. Aortic lesions have the disadvantage that they occur either in young men subjected to the influences of hard work, alcohol, etc., or in older men of the arteriosclerotic type. The greater frequency of coronary arterial accidents, angina, etc., is a factor. The favorable influence of a moderate stenosis in case of extensive insufficiency at either the aortic or the mitral valve should be noted.

Children have the great disadvantage that the valves have not yet attained their full growth, and the lesion prevents their perfect development. Many fail at the time of the increased growth at puberty. In the absence of any other explanation of the failure of compensation in a child, it is fairly safe to assume an adherent pericarditis. It is perhaps the most serious feature of cardiac disease in children.

An occasional exception to the general rule of bad prognosis in children may be observed. I treated a boy of 7 years for rheumatic endocarditis, with broken compensation, dyspnea, edema, dilatation of the heart, and such severe epistaxis that a specialist was first called by the family on account of it. He responded well to treatment; and wheel-riding, which he had followed inordinately for such a little fellow, was forbidden. He is now a man of 25 years, of full stature, and with perfect compensation, but with a loud systolic murmur, wide transverse enlargement of the heart, and accentuated pulmonic second sound. He has meanwhile had a severe recurrence of acute articular rheumatism. For one such case a score probably fail to pass puberty. I have mentioned a physician who worked hard for more than 45 years after the development of mitral regurgitation following acute rheumatism. He took extraordinarily good care of himself, however, stopping half way up every

stairway to recover from his dyspnea. He finally died at the age of 78 of cerebral hemorrhage.

Many cases die prematurely from foolish overexertion. One boy who was doing perfectly well overstrained his heart by riding against a head wind in a bicycle club run, and never got out of bed again. A man in the thirties with mitral stenosis was cautioned, as I have advised many such, never to get into a position where he might be forced to overexert himself. The next week he drove a restless colt, which ran away. Unable to stop him, he merely held him in the road for miles. Death shortly resulted. The probability of the proper following of advice given is therefore to be considered in prognosis.

## 6. CONGENITAL AFFECTIONS OF THE HEART

These occur either from failure of proper development of the organ or as the result of intra-uterine endocarditis. The tendency of the inflammation to attack imperfectly formed valves is well recognized. Absence of the heart, and other conditions incompatible with life are of interest to the embryologist. The table given by Holt shows something of the frequency of different congenital lesions:

### FREQUENCY OF CONGENITAL LESIONS

Defect in the ventricular septum.....	149 cases; only lesion in 5 cases
Defect in the auricular septum or patent foramen ovale.....	126 " " " " 9 "
Pulmonic stenosis or atresia.....	108 " " " " 6 "
Patent ductus arteriosus .....	68 " " " " 3 "
Abnormalities in the origin of the great vessels .....	45 " " " " 0 "
Pulmonic insufficiency .....	17 " " " " 0 "
Tricuspid insufficiency .....	6 " " " " 0 "
Tricuspid stenosis or atresia.....	3 " " " " 0 "
Mitral insufficiency .....	1 case; " " " " 0 "
Mitral stenosis or atresia.....	6 cases; " " " " 0 "
Aortic insufficiency .....	1 case; " " " " 0 "
Aortic stenosis or atresia.....	6 cases; " " " " 0 "
Transposition of the heart.....	2 " " " " 0 "
Ectocardia .....	1 case; " " " " 1 "

## THE MOST FREQUENTLY ASSOCIATED LESIONS

Pulmonic stenosis with defect in the ventricular septum.....	92 cases; only lesion in 20 cases
Pulmonic stenosis with defect in the auricular septum....	52 " " " " 8 "
Defects in both septa.....	82 " " " " 17 "
Pulmonic stenosis and defects in both septa..	36 " " " " 21 "

The most important lesion is congenital pulmonic stenosis. The subjects of this affection may grow to adult life. Defects in the ventricular septum are often associated with it. Next in importance is patent ductus arteriosus. Coarctation of the aorta, a narrowing just beyond the entrance of the duct of Botalli, is an interesting congenital anomaly.

**Symptoms of Congenital Heart Disease.**—The most frequent and striking one is cyanosis, and most of the "blue babies" are of this type. The lips, fingers, and toes are most affected, but the whole skin is bluish in certain cases. It is much exaggerated upon exertion, and the frequent coughing from which these children suffer in winter gives abundant opportunity to observe this increase. The clubbing of the fingers and toes is noticeable. In one such case, which I have just seen with Dr. M. J. Krohn, its healthy twin sister was eight inches taller at six years of age. Dyspnea is oftentimes as marked a symptom as the cyanosis. The usual polycythemia of cyanosis is present. Recurrent laryngeal paralysis occasionally results from pressure of a dilated ductus arteriosus.

**Physical Examination.**—The common finding in congenital heart disease is a systolic murmur in the pulmonic region, since stenosis of the pulmonary artery is so predominantly the usual lesion, while patent ductus arteriosus gives a similar murmur. In general no very accurate diagnosis can be made, and especially because many cases present no definite physical signs, aside from enlargement and cyanosis. I have reported a case in which a systolic murmur was detected *in gravida*, and heard easily after birth. From its location at the base of the heart, and its disappearance during the early weeks of life, it may have been connected with the ductus arteriosus. I later saw a similar case, reported by Dr. H. G. Wetherill.

The frequency of cardiac anomalies in situs viscerum inversus has long been recognized. I have observed two cases of congenital heart disease in boys with complete transposition, one having so large a heart that the dullness could be detected in the right back. Both presented such signs and symptoms as would have led to a diagnosis of mitral regurgitation, excepting for the anomalous position of the various valves. No autopsies were secured. One died at about 20 and the other was doing badly when last heard from at 10 years of age.

**Prognosis.**—Most of the cases of congenital heart disease die during the first week, and perhaps three-fourths of them in their first two years. Of those living longer a majority show pulmonic stenosis as the chief lesion.

### **7. RUPTURE OF THE HEART**

This takes place most frequently in conditions of myocardial degeneration resulting from coronary arterial disease, and most often in the anterior wall of the left ventricle. Death is sudden, following exertion in most cases.

Diagnosis is impossible.

### **8. ANEURISM OF THE HEART AND VALVES**

Aneurism of the heart commonly results from the progressive stretching of a portion of the wall weakened by myocardial degeneration, by a gumma, or injury. The left ventricle is oftenest affected. Rupture is not infrequent. The symptoms are not sufficiently definite to permit diagnosis in most cases. Diagnosis with the fluoroscope will perhaps be accomplished in future, in certain well-marked cases.

Aneurism of the valves is generally the result of an acute destructive process in severe endocarditis. It is of interest because of the rupture of the valve which occasionally results.

### **9. ANGINA PECTORIS**

This is a disease characterized by paroxysms of severe pain about the heart, and by arterial and myocardial changes.

**Etiology.**—The disease is about five times as frequent in males as in females. It is most prevalent in the fourth, fifth, and sixth decades, and is more frequently found later than earlier. It is found in those conditions in which the nutrition of the myocardium suffers, generally because of arteriosclerosis in the coronary arteries, but probably at times because of vasomotor changes without organic basis. It is especially seen in those subjected to nervous strain, and in whom the factors tending to the production of arteriosclerosis are also operative. Of these syphilis is most important, especially in the younger patients. High blood pressure associated with arterial changes and frequently with the overuse of tobacco is very commonly present. The affection is vastly more frequent than formerly. Rush stated in his lectures that he has seen but a single case and knew of but one other.

**Pathology.**—The usual findings in angina pectoris are those of extensive atheromatous changes in the coronary arteries. The vessels may be generally calcified or narrowed only near the entrance, or a single one may have a single point of partial obstruction, which has been blocked by embolism or thrombosis. The essential feature is that some change has occurred in the arteries to prevent the myocardium from receiving the absolutely constant supply of blood so imperative to a muscle which must continue its function uninterruptedly if life is to continue. In intermittent claudication one may suspend the effort to walk until the leg muscles are relieved by a fresh supply of blood through the sclerotic arteries, but in angina the myocardium must have its continuous supply, or death results. Extensive atheromatous changes in the aorta are often seen, especially in the younger subjects with syphilis. In rare cases no definite pathology has been determined.

The exact mechanism of the production of the agonizing pain of the disease has not yet been agreed upon. Analogy leads one to consider the theory of intermittent claudication as very acceptable. Mackenzie believes the attacks to be due to exhaustion of the function of contractility of the cardiac muscles due to struggling against too great a load, whether that load be absolutely great, or only relatively so as compared with the strength of the muscle. Relief from the

attacks as soon as the mitral valve gives way in the frequent dilatation of the heart in this affection is thus explained as being due to unloading of the left ventricle. Of the immediate exciting causes, overexertion, generally physical, but often mental, is the most frequent. By walking up hill, the patients may come close to the line of attack, may then rest and note the passing away of the symptoms, and bring them back or even instigate a full attack by further exertion. The frequency of the attacks after public speaking, outbreaks of anger, etc., is well recognized, and a few are associated with attacks of indigestion.

**Symptoms.**—In typical cases the patient is suddenly seized with pain believed to surpass in degree any other known, generally during physical or mental exertion. It is felt as a constrictive agony in the region of the heart, and generally radiates to the left side of the neck and down the left arm to the fingers. The patient holds himself still, and the ashen countenance, the look of agonizing pain, and the expression in the countenance of the fear of instant death make up a clinical picture seen in no other disease. An asthmatic type of wheezing with dyspnea may be present, and even the condition of acute emphysema. Sweating may shortly occur, and after a fraction of a minute or several minutes the attack subsides, syncope sometimes preceding the subsidence. After the attack free belching may occur. Mackenzie believes that this is air swallowed during the attack. The distribution of the pain varies greatly. I have once or twice noticed the distribution to the right arm as much as to the left, and in one case the lobe of the left ear was the seat of pain described as agonizing. A recent patient 62 years of age complained of little excepting dyspnea and an excruciating pain in the roof of the mouth. Inquiry showed that the attacks always started in the cardiac region after exertion, and extended to the left arm and hand, but the palatal pain overshadowed everything else. Radiation to the back and kidney is occasionally noted.

In the severest type the patient drops dead in the height of the attack. If the opportunity be afforded for examination of the pulse it may be found regular, but generally with increase of tension, even to 300 mm. of mercury. Hyperesthesia of the region



most affected by the pain may be found after the subsidence of the attack. Free urination is common at this time. Many patients live in constant dread of recurrence of the paroxysm. In certain cases the attacks recur, generally in milder form, so frequently as to make really a prolonged anginal attack of several hours' duration.

Although the pain is normally in the area of distribution of the first to the fourth left dorsal nerves, inclusive, it is not unusual for the seventh and eighth cervical and the fifth to the ninth dorsal segments to be involved, and even the nerves of the right side. The so-called *angina abdominis* is thus explained. Allbutt believes it to be due to dilatation of the abdominal aorta. No arteriosclerotic patient should be treated for abdominal disease, as gall-stone colic, gastralgic attacks, etc., without careful consideration of the possibility of anginal origin. Pain in the testes and the left leg has been reported in certain cases as the initiatory feature. Osler reports monoplegia, hemiplegia, and aphasia in rare cases. An acute pulmonary edema may occur.

Many milder types of *angina pectoris* are noted. The symptoms may be merely those of distress and a feeling of tension beneath the sternum, associated with physical or mental effort, and soon disappearing. Such symptoms should be a warning to the patient to get out from under his load by taking a vacation, when the distress may utterly disappear. In the cases which are found post-mortem to have no very definite pathological basis, the attacks are generally of a mild type, and presumably depend upon temporary myocardial starvation from vascular spasm. These attacks occur as a rule in younger patients; and the overuse of tea, coffee, tobacco, and alcohol is frequently admitted. The numbness in the extremities, with coldness and pallor, and the faint feeling testify to the vasomotor disturbance which constitutes the basis of the attacks. Palpitation may be present. Such attacks are rarely fatal.

**Course.**—If the heart dilates and relative mitral insufficiency be established, the attacks commonly cease, but the patient has the edema and other signs of failing circulation. I have seen a physician who recognized the full significance of the edema following mitral leakage rejoice because of the relief felt at the assurance that

he would probably have no further anginal attacks, his fear of them entirely overshadowing his fear of death. A few cases die tragically in the first attack, while others live through many paroxysms, and die perhaps of intercurrent disease.

**Diagnosis.**—The character of the pain in the genuine attacks, with the associated arterial changes, high pressure and frequent history of syphilis, leaves little room for doubt. The attacks of the vasomotor type, which may even occur in hysterical women, are not accompanied with the increased vascular tension nor degenerative arterial condition, and yet the pain is obviously severe. The abdominal form presents the features of the true angina, excepting for the addition of the abdominal pain, sometimes its substitution for the chest pain. If it be borne in mind that angina pectoris may present most anomalous features as to the pain distribution, no error is likely to occur.

**Prognosis.**—This is best in the attacks of vasomotor type, many of these patients recovering. The young men with specific history are often amenable to cure by vigorous treatment. In the older cases with definite arterial changes, any attack may prove fatal. High blood tension is perhaps the worst single prognostic feature.

### CORONARY OBSTRUCTION

Herrick classifies the cases as follows:

One group will include cases in which death is sudden, seemingly instantaneous and perhaps painless. Krehl has emphasized the peculiarities of the sudden death of this type, the lack of terminal respiratory agony, of distortion of the features, of muscular contractions.

A second group includes those cases in which the attack is anginal, the pain severe, the shock profound and death follows in a few minutes or several minutes at the most.

In a third group may be placed non-fatal cases with mild symptoms. Slight anginal attacks without the ordinary causes (such as walking), perhaps some of the stitch pains in the precordia, may well be due to obstruction of small coronary twigs. Such an interpretation of these phenomena is, however, only a surmise based on the fact that other causes for the pains are lacking and that the patchy fibrosis of the myocardium that is later found at autopsy may have originated in obstruction of the sclerotic vessels; and such obstruction in small vessels may well have produced symptoms differing

chiefly in degree from those caused by obstruction of larger arteries of the heart.

In a fourth group are the cases in which the symptoms are severe, are distinctive enough to enable them to be recognized as cardiac, and in which the accident is usually fatal, but not immediately, and perhaps not necessarily so.

## 10. ANEURISM

In this disease the coats of the artery yield, because of injury or degeneration, to the blood pressure, and form a more or less circumscribed dilatation of the arterial wall. In true aneurism the walls are formed by one or more coats of the artery. In false aneurism the coats are ruptured and blood escapes and forms a hematoma. In arteriovenous aneurism, once common as a result of blood letting, a communication exists between artery and vein, sometimes a definite sac being present. In fusiform aneurism there exists a spindle-shaped dilatation of the artery, without the definite saculation, which is more characteristic of aneurism in general. It is also called "dilatation aneurism." If extensive in the distribution of an artery and its branches, it is called cirroid aneurism. By dissecting aneurism is meant one that separates the coats of the artery, at times forming a new passageway for the blood. A sacculated aneurism is one bulging sharply from the wall, involving any of the coats and making a definite sac, as is not infrequent in the first portion of the arch.

**Etiology.**—Resulting as it does from those causes which weaken the walls of the artery, and at the same time often tend to raise the blood pressure, it is natural that males, in whom arteriosclerosis predominates, should be more often affected, the ratio being about five males to one female. As in arteriosclerosis, syphilis and laborious occupations are notable factors, both being operative in many of the cases seen in the colored race, and in soldiers, stevedores, forgers, and others in whom aneurism is frequently found. The affection is most common in the fourth decade, the fifth coming next in frequency. The constriction of the equipment carried by soldiers is thought to be a contributing cause by some English army surgeons.

In the cases resulting from trauma, severe exertion, etc., there is generally a preceding arteriosclerotic weakening of the wall. The

rôle of syphilis in producing this weakening is known to be even greater than was formerly thought, since the Wassermann reaction is found in a very large proportion of all cases. Severe strain, as in a violent lift, is often accountable for the development of aneurism.

Aneurism is, as a rule, a development rather early in arteriosclerosis, the subject being at the height of his muscular strength in many cases. A local giving way of the intima over a weakened spot in the media results in the production of a sacculated aneurism. The general weakening of the wall creating the diffuse or fusiform aneurism is more common in subjects more advanced in age and with general atheroma. In rare cases multiple aneurisms of mycotic origin are found, and damage to the coats from embolism of the vaso vasorum may be responsible. Thoracic aneurism, the variety of most interest to the medical man, was present in about one-fourth of the 501 cases collected by Crisp, and aneurism of the abdominal aorta, in about one-eighth. The others were scattered, the popliteal artery exceeding the thoracic aorta in frequency of involvement. Sixty per cent. of all aneurisms of the arch affect the ascending portion.

#### A. ANEURISM OF THE THORACIC AORTA

In order of frequency, the aneurisms of the aorta are the sacculated, the dilatation, and the dissecting varieties.

##### SACCULATED ANEURISM

The sacculated aneurism of the aorta commonly affects the arch, the first portion most frequently. Here the tumor grows forward and presents at the second or third interspace as a pulsating tumor, frequently eroding through the ribs. It may push over more to the right and enter the pleural sac, or, springing from the left side of the vessel, push over to the region of the left clavicle and first and second ribs. It is so near the surface that its pulsations are obvious, and the through-and-through pulsation may be very striking. As it grows, compression of the right sub-clavian vein or the vena cava superior may occur, with enlargement of the right arm in the former

instance, or the head and neck in the latter. It is in this type that right recurrent paralysis is found from pressure upon the nerve as it winds under the right sub-clavian artery. The growth of the sac confined by the bony chest wall leads to downward displacement of the heart, often to the left, and even to obstruction of the inferior vena cava with obstructive phenomena in the lower limbs. If the transverse arch be the seat, the symptoms are those of compression of the many delicate structures of that region, with less in the way of external signs, though bulging may appear in the right upper sternal region, or the severe boring pain characteristic of pressure upon the spine may appear. From its proximity to the esophagus, left recurrent laryngeal nerve, trachea, and main bronchi, innominate arteries and other branches of the arch, thoracic duct, and sympathetic nerve ganglia, there may be observed dysphagia, aphonia, jerky respiration from the affection of the trachea by the pulsations, attacks of severe dyspnea, cough, and expectoration from the destruction of lung incident to permanent compression of the left bronchus, extension of the aneurismal sac to the innominate or other arteries, serious interference with nutrition, or even chylothorax, and finally, from the sympathetic involvement, flushing with pupillary contraction on one side of the face and sometimes sweating, or pallor, with dilatation of the pupil. I once saw with Dr. Rover a patient with the signs of paralysis of the sympathetic, flushing and contracted pupil, on one side, and those of irritation, pallor, and dilated pupil upon the other, at the same time. The so-called Babinski syndrome refers to the presence with aneurism of tabetic features, of which the Argyll-Robertson pupils or unequal pupils are of especial interest in this connection. In the descending portion of the arch the tumor projects backward and compresses the spine, or perhaps slightly to the left, and sets up an eroding process affecting the third to the sixth ribs, frequently with the burning pain of a pressure neuritis running around the left breast. The steady, boring, terrible pain of erosion of the spine, often little relieved by medicine, should always suggest erosion by aneurism. Paraplegia eventually occurs in certain of these cases. Dysphagia and bronchial obstruction are not uncommon. This is the type in which a tumor projecting through

the ribs explains the reason for left-sided intercostal neuritis mentioned. In a few instances the aneurism affects the thoracic aorta below the descending portion of the arch. It is more prone to be silent, and is often overlooked. Severe erosive pain, pleural or pulmonary symptoms, and dysphagia may be present, and the tumor may show externally. Aneurism of the region of the sinuses of Valsalva is rarely diagnosed clinically, though not rare post mortem, often having caused sudden death. Its striking features lie in its occurrence in young syphilitic subjects, with frequent involvement of the aortic ring, with perhaps aortic insufficiency, and anginal attacks in certain cases, its latency, and its tendency to cause sudden death by perforation. The aneurism of the ascending portion may perforate into the vena cava, producing extreme venous engorgement over the chest, with dyspnea, and the "humming-top" murmur, with sharp systolic accentuation. The suddenness of the onset of so serious a condition in one not known perhaps to be ill is very striking. I have observed two such cases in the Denver City Hospital. A similar perforation into the pulmonary artery is occasionally found with somewhat similar symptoms, as in a case to be quoted later. In the aneurism of the ascending arch, death may occur from rupture externally, or into the right pleura, pericardium or vena cava. In the transverse aneurism it is not infrequently due to bursting into the trachea or left bronchus, or to "aneurismal phthisis" incident to the pressure upon the left bronchus. Erosion of the spine and paraplegia may bring about the fatal result. In the descending type, lung and pleural complications, often with hemorrhage and involvement of the spine are modes of death.

**Symptoms.**—The absence of symptoms in certain cases of aneurism is notorious, many cases dying suddenly without a suspicion of the cause. Pain is the most constant symptom. It may be paroxysmal and occasionally anginal in character, and in general of anginal distribution. The ominous pain coming from erosion of bone by the tumor, of which the patient complains bitterly week after week, is so striking that in any chest case a pain of this nature should cause careful examination for aneurism. The pressure neuritis when the intercostal nerves are involved has been mentioned.

The frequent sensitiveness over painful areas should be noted. Dyspnea is common as a result of laryngeal disturbance, pressure upon the lung in general, damage to one lung through pressure upon the main bronchus, or, worst of all, from pressure upon the trachea with collapse of the tube. Stridor is often present. The hoarseness of recurrent paralysis is a very suggestive sign, and the "goose cough" as well.

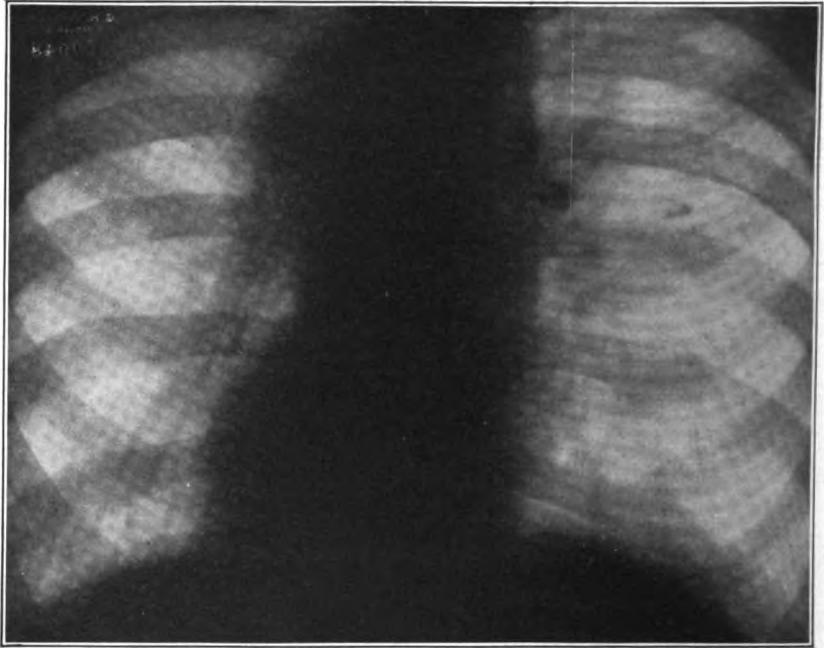


FIG. 41.—POSTERO-ANTERIOR VIEW OF ANEURISM OF ARCH OF THE AORTA. (Dr. G. H. Stover.)

The laryngeal signs and symptoms are so important that I quote from Dr. Levy's remarks upon this subject at a clinic at the Denver City Hospital at which I showed four cases of aneurism of the transverse portion of the arch illustrating nearly all the interesting laryngeal features of the disease.

The first laryngeal symptom of aneurism refers to the respiratory function of the larynx. There is a moderate amount of dyspnea, marked, however, upon exertion. During rest and tranquil respiration there may be

detected a slightly noisy inspiration. This symptom is due to a paralysis of the respiratory muscles of the larynx, namely, the posterior crico-arytenoids. These muscles abduct the vocal bands, thus enlarging the chink of the glottis and permitting the air to pass freely through the larynx. When these muscles are paralyzed upon one side, the adductors having no antagonist in their action hold the vocal band of that side in the median line. This fact accounts for the almost normal voice in the early stage of recurrent paralysis. The reason that the abductors are first affected is a much mooted question, but the majority of laryngologists have accepted the explanation of Sir Felix Semon, which he formulated a few years ago in what is known as Semon's law. This author believes that there is a "difference in the biological composition of the laryngeal muscles and nerves," which makes the abductors more susceptible and causes the symptoms of slight pressure to manifest themselves in the nature of interference with the respiratory function.

In a certain proportion of cases the stage during which the abductors alone are involved is extremely short or does not exist at all. This is particularly true of cases in which the enlargement of the aorta occurs itself suddenly or is principally in the anterior wall of the aorta. Here the pressure is made almost complete at once and involves the pneumogastric as well as its branch, the inferior laryngeal. The symptoms of increased pressure are those of complete paralysis of the recurrent laryngeal, namely, the loss of motion of all of the intrinsic muscles of the larynx except the cricothyroid. These muscles include the adductors, the principal of which are the lateral crico-arytenoid, the internal thyro-arytenoid, the tensors, and the arytenoid. It is obvious, therefore, that when the nerve is completely paralyzed and both abductors and adductors fail to act, that the vocal bands be neither abducted nor adducted, nor will they be made tense, but will rest in a position midway between abduction and adduction and be lax. This position of the vocal bands is what is known as the cadaveric position.

The dyspnea in one case may be explained either by pressure on the trachea or bronchi, or it may be due to so pronounced a pressure upon the pneumogastric that the irritation is transmitted to the center in the medulla, then to the opposite side, causing spasm of the opposite abductors. In this way pressure upon one pneumogastric will cause marked bilateral manifestations, especially giving rise to laryngeal dyspnea.

It is important to recognize whether the dyspnea be due to this cause or to pressure upon the trachea or bronchi. If due to the former, tracheotomy will relieve the urgent distress, and if due to the latter, opening into the trachea will be of no avail.

The voice while aphonic from the beginning may gradually improve. This is not an uncommon manifestation, but should not be construed to mean improvement in the actual condition. One vocal band may be completely paralyzed, remaining midway between abduction and adduction, while the other in course of time takes on compensatory action and passes beyond the median line to closely approximate the paralyzed band. When



this exaggeration of the unparalyzed vocal band takes place, the voice begins to return, and while the patient will never have a normal voice, he will nevertheless be able to phonate better than early in the affection. There is marked distress during prolonged phonation due to what might be called respiratory loss. The chink of the glottis being larger than normal every act of phonation permits an increased loss of breath and the patient becomes rapidly exhausted in an attempt to use his voice.\*

Dysphagia has been mentioned and is generally due to mechanical interference, sometimes to spasm. The sound should not be passed for fear of perforation. The pupillary symptoms are discussed elsewhere. Hemorrhage may be merely trivial, from bleeding granulations, or severe, as from perforation into the pleural cavity, pericardium, bronchus, trachea, or externally. The left bronchus is a favorite site, and death in such cases is commonly instantaneous. The bleeding may be checked by the floating of a "patch" of fibrin over the orifice, and the patient may live even for years.

**Physical Examination.**—**INSPECTION.**—The patient's eyes should be closely examined, as there may be pupillary signs due to aneurism, through the influence of the sympathetic, or, nearly as important, difference in size of the pupils, irregular pupils, or Argyll-Robertson pupils, suggesting a syphilitic history. Any middle-aged man with a dilated pupil, a husky voice and chest symptoms should be suspected of having aneurism.

The face is often congested, and may be definitely edematous if the superior cava be compressed. The neck is occasionally doubled in size under these circumstances, with much-dilated veins. The head and neck may move with expansion of the sac. The trachea and larynx may be displaced laterally, and close inspection may show a visible tracheal tug. In the upper chest and neck there may be local pulsation. This is often best seen if the light be permitted to fall sidewise on the chest. The enlarged veins of the chest and of one or both arms are noticeable if venous obstruction exist, and one arm may be visibly larger than the other. The valves may bulge. Clubbing of the nails or nutritional changes in the skin of the hand or maceration from sweating may be present. The pulsation may involve the whole upper chest, or be limited to the tissues lifted by the

\* *Boston Medical and Surgical Journal*, January 17, 1907.

aneurism, or represent the sac itself close under the skin. In case perforation of the chest wall has occurred the skin over the aneurism may be congested and edematous, or even perforated. The heart may be seen to beat in an abnormal position, most frequently downward and to the left.

**PALPATION.**—By this we may note the shock of impact of the expanding sac, and the expansile character of the tumor. The diastolic shock transmitted into the tumor is one of the most reliable of aneurismal signs, and excludes a solid tumor if plainly felt. Infrequently a thrill, which may be systolic, and occasionally diastolic or double, may be found. By placing one hand in front and the other behind the chest, the through and through expansion of the chest may be detected, and it constitutes very reliable evidence of aneurism. If the head be slightly elevated and the finger and thumb be placed under the cricoid cartilage, tracheal tugging may be found in the majority of cases, due to the expansion of a sac directed in such a way as to push downward upon a main bronchus and thus dislocate the trachea and larynx downward, or to direct movement of the trachea through adhesions. Although a slight pulsation or indefinite tug counts for little or nothing, the decisive “tug” like that of a heavy fish on the line, is of the utmost value in the diagnosis of aneurism. The slighter tug, which is found on deep inspiration in cases of old left pleuritic adhesions and other conditions, should not be taken too seriously. In case the diastolic shock be not felt over the tumor, it may in certain cases be felt through the fingers used in testing for tracheal tugging as a distinct shock at the time of closure of the aortic valves, and therefore instantly after the tug. I have never known it to be present in mediastinal tumor. In a report by Dr. Leonard Freeman and myself of two cases of aortic aneurism, successfully wired, I have noted that this phenomenon disappeared as the aneurism solidified, the tracheal tug remaining apparent for several weeks thereafter. *See Am. Jour. Med. Sc., Dec., 1901.*)

The simultaneous palpation of the two radial pulses is important in the diagnosis of aneurism, as they may be unequal, asynchronous, or one may be absent. The left radial has returned after

being absent a year in a case now in the Denver City Hospital. The interference with the right pulse in general suggests that the aneurism involves a part near the innominate artery; that of the left, that it compromises the arch beyond the innominate; while an aneurism beyond the origin of the left subclavian artery would scarcely affect either. Since, however, pressure, dislocation of organs, and perhaps other factors may influence this, it may be safest to say that inequality



FIG. 42.—ATHEROMA AND FUSIFORM DILATATION OF THE THORACIC AORTA. Postero-anterior view. (Dr. G. H. Stover.)

of the pulses or delay or absence of one is a very reliable sign of aneurism. The abdominal aorta may be without pulsation in a very large thoracic aneurism, because of the effect of the expansile sac in taking up the pulsations, as does the "dome" on the steam fire engine, so that a continuous stream is sent out.

**PERCUSSION.**—Percussion shows flatness over the tumor if it be near the surface. The compression of lung over an aneurism may give dulness before pulsation or bruit can be detected. In case

of left-sided intercostal neuralgia (pressure neuritis), it is well to percuss carefully down the left side of the spine, as dulness may signify the presence of a deep aneurism. I have located one in this way months before the pulsation became apparent.

**AUSCULTATION.**—No murmur may be heard since the tumor may contain fibrin to such an extent that none is produced. In general the systolic bruit is heard, carried along in the direction of the blood stream. Valvular murmurs may also be present, especially those of aortic regurgitation and relative mitral insufficiency. The former is of some direct import, since the valves are not infrequently distorted by the arteriosclerotic process, or by the tumor. The aortic second sound is commonly sharply accentuated. The whirring murmur in case the aorta has burst into the vena cava superior has been mentioned. I have seen an identical murmur with systolic intensification in perforation into the pulmonary artery.\* Feeble respiratory murmur on one side is commonly found if the bronchus be compressed.

The blood pressure very often varies in the two radial arteries, and any considerable difference is suggestive of aneurism. The systemic blood pressure is not notably affected in most cases.

\* *Boston Med. and Surg. Journal*, Jan. 17, 1907.

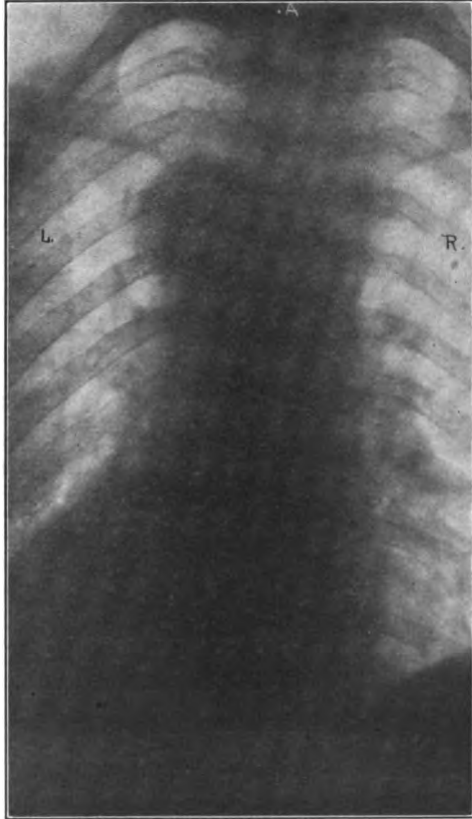


FIG. 43.—ANEURISM OF AORTIC ARCH DISPLACING TRACHEA. (Dr. G. H. Stover.)

The X-ray examination of the subject should never be neglected, and the properly interpreted plates in general furnish more reliable evidence than the fluoroscope, even though pulsations may be detected by the latter. Aortic aneurisms are occasionally discovered by accident when examining the chest for other purposes, as in a recent case in the Denver City Hospital.

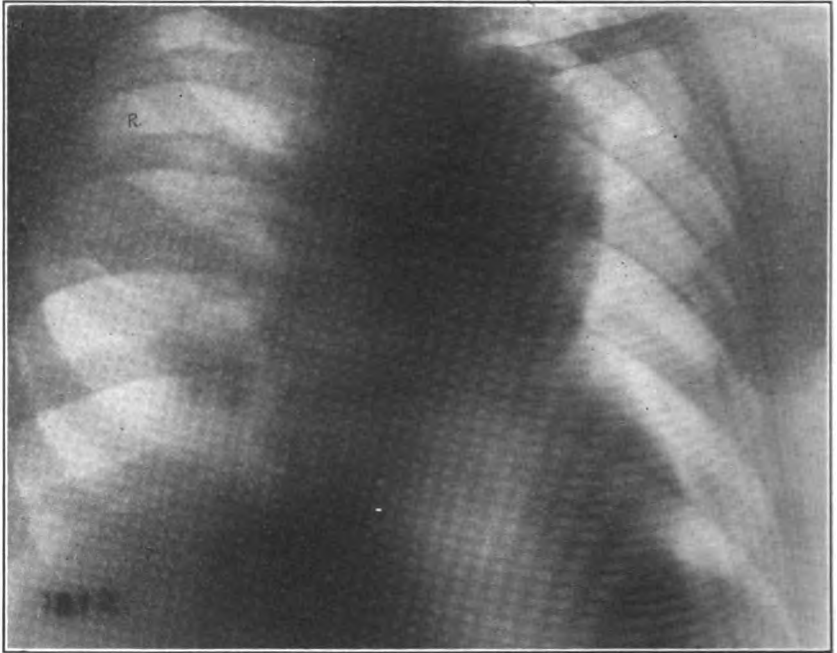


FIG. 44.—ANEURISM OF THE DESCENDING PORTION OF THE AORTA.  
(Dr. G. H. Stover.)

#### DILATATION ANEURISM

A more or less uniform dilatation of the aorta, perhaps of the ascending portion of the arch, at other times of the whole thoracic portion, is recognized under this title. It is frequently associated with two features of special importance in the etiology, namely, arteriosclerosis and aortic regurgitation.

**Symptoms.**—So slight are the symptoms in most cases that the frequency of the condition was not recognized until the common use

of the Röntgen ray. Symptoms such as are commonly associated with advanced arteriosclerosis and aortic regurgitation are present, and anginal symptoms and those of failing heart strength are often found. As may be inferred, most of the cases occur at the arteriosclerotic age.

**Physical Examination.**—The first finding to suggest the condition is the wide area of dullness in the upper sternal region, which commonly leads to the Röntgen-ray examination. This shows a wide shadow running from the heart upward. The filling of so large an aorta during systole makes relatively so little difference that pulsation may not be visible with the fluoroscope. In the upper sternal region, at the notch and the upper interspaces on each side, palpation may show pulsation in extreme cases. Added to this are the auscultatory signs of aortic leakage, in certain cases, and aortic accentuation in others. Thrill, systolic or diastolic, may be present. Although the signs of arteriosclerosis are very prominent, the blood pressure is generally not raised above the normal. In certain cases the through-and-through pulsation is apparent.

#### DISSECTING ANEURISM

This type begins with a sudden severe pain and shock incident to the rupture of the inner coats of the aorta at some point previously weakened by atheromatous processes. If death be not immediate, it may occur some hours or days later when the remaining support gives way. In a few cases the outer coats hold and the aneurism dissects its way for a variable distance, even down to the femoral arteries. This extraordinary condition may last for years, a new intima developing in the false passage.

**Diagnosis.**—The presence of a pulsating tumor with such signs and symptoms as we have mentioned may generally be accepted as positive evidence of aortic aneurism, but certain exceptions must be noted. First, the aneurism may be present and may be absolutely devoid of pulsation from being filled almost solidly with fibrin, as in a case I once saw with Dr. Sewall. The pulsation may be due to a very vascular tumor. It may be the dynamic pulsation of a

very expansible aorta, or the violent pulsation in case of aortic regurgitation. The position of the heart should be established before charging a pulsation in the chest to any other condition. Pulsating pleurisy generally occurs upon the left side, and the febrile course and extensive respiratory signs, with the help of the Röntgen ray and aspiration, should settle the question. The great difficulty comes in the case of solid tumor. Aneurism and cancer may occur together, as in a case I have mentioned elsewhere (diseases of the mediastinum). In general the pulsatile features, accentuated aortic second sound, tracheal tug, diastolic shock, either at the site of the tumor or in the trachea, as described, and the general symptomatology suffice, and with the help of the X-ray can scarcely leave one in doubt. The absence of glandular involvement and of cachexia speak strongly for aneurism. In doubtful cases it is always safer to assume that aneurism is present if the left recurrent nerve is involved.

In case wiring of the aneurism is to be considered, a diagnosis of the position and character of the sac is the first essential. The X-ray is now of much assistance. I quote the conclusions of Dr. Leonard Freeman and myself in this matter, taken from our report of two cases successfully wired:

To obtain the best results it seems evident that the cases should be carefully selected. The aneurism should be sacculated, not fusiform; without too large an opening, and with but one compartment. It should not have progressed too far toward rupture, and the patient should have sufficient vitality to enable him to live through the tedious process of cure. It is scarcely necessary to state that the operation is practically confined to aneurisms not amenable to ordinary surgical procedures, i.e., aneurisms of the abdominal aorta, the thoracic aorta, the innominate artery, etc., the first named requiring a preliminary cœliotomy.

Surgical measures are necessarily limited to those aneurisms accessible from the surface, either directly or after opening the abdominal wall. I speak to-day chiefly of those springing from the anterior or lateral surfaces of the ascending portion of the arch of the aorta and of such sacculated character that clotting of their contents would not shut off the direct current through the aorta. A very small percentage of the aneurisms of the arch are of this nature, but, where the diagnosis can be made, the gloomy prognosis of medical treatment should lead us to offer surgical relief.

In a general way, the aneurisms of the ascending portion of the arch, eligible for surgical treatment must be those presenting chiefly the signs of

growth anteriorly or laterally. These signs are entirely consistent with sacculatation of the aneurism. Even though sacculated, if the tumor project from the posterior wall of the ascending aorta it must be looked upon as ineligible for such treatment, not only because of its inaccessibility, but because of the difficulties in the way of the diagnosis.

The signs and symptoms found in the aneurisms mentioned as being suitable for treatment are in general those that indicate growth toward the surface, with involvement of the parietes, rather than toward the centre of the chest, with pressure upon internal organs. Thus we have pressure upon the ribs or sternum, with consequent neuritic pain, bulging of the bony parts, a pulsatile and expansile tumor, dulness, diastolic shock, and perhaps auscultatory signs or the inflammation of the skin which precedes ulceration, as in one of our cases.

It is not common in these cases to have dysphagia or pressure upon the trachea or bronchi. Hoarseness or aphonia from pressure on the recurrent laryngeal nerve, pupillary signs from involvement of the sympathetic, and tracheal tugging and diastolic shock, however, may all be present, as in the first case to be reported, without contraindicating the attempt or preventing a successful issue. Inequality of pulse in the two radials, with asynchronism, may occur, owing either to pressure upon or distortion of the orifice of the innominate or subclavian, or to the delay of the pulse-wave in the large sac.

It is quite possible that we may, in selected cases, disregard all of the signs of intrathoracic pressure, for they may come from the pushing back of the vessel by an aneurism bulging anteriorly, as was probably the case in the instance just mentioned, where even slight dysphagia existed.

**Prognosis.**—Aside from cure by wiring and the spontaneous healing of the small sacculated aneurisms at the base of the aorta, the outlook is bad. Yet tumors of the first portion and even dissecting aneurism may be compatible with life for many years in rare cases.

## B. ANEURISM OF THE ABDOMINAL AORTA

This is much less frequent than that of the thoracic portion, the ratio being at least ten to one in favor of the latter. The general etiology and pathology are practically the same for both. The most common location is below the diaphragm and the shape may vary as in the thoracic type. Severe pain, especially in the bone-eroding type, is frequent, and it generally signifies involvement of the spine. Various digestive symptoms, especially vomiting, may be present. Paraplegia is a frequent result of the spinal pressure. The tumor may be inaccessible to palpation because of its high position under the



diaphragm, or may be felt in the left abdomen or left loin. A deep abdominal swelling may develop from rupture and formation of a false sac posteriorly.

**Diagnosis.**—This is the region of the “aneurism of students,” as the dynamic pulsation of the abdominal aorta in thin neurotic subjects is called. No diagnosis should be based upon pulsation, unless, as Osler well says, there is “a definite tumor, which can be grasped and which has a definite pulsation.” Lifting of a tumor of any kind lying upon the aorta is common, but should cause no confusion. The Röntgen ray should settle the diagnosis in a doubtful case. The outlook is bad, death usually resulting from paraplegia, from blocking of the vessel, or rupture.

**Other Aneurisms in the Abdomen.**—Aneurisms are occasionally found in the splenic, hepatic, superior mesenteric and renal arteries. They create pain and pulsation but the diagnosis can scarcely be made excepting by exploration, unless the X-ray plate be of service.

### C. ANEURISM OF THE PULMONARY ARTERIES

This is rare in the trunk but is occasionally found in the branches. Pulmonary hemorrhage is not infrequent in tuberculosis from rupture of a small aneurism of an arterial branch in a cavity.

### D. ARTERIOVENOUS ANEURISM

The term varicose aneurism is applied when a sac exists between the artery and a vein; aneurismal varix when no sac exists. They are both generally traumatic in origin. The communication of the aorta with the superior cava has been mentioned in the proper section. The abdominal aorta occasionally communicates with the inferior cava. The limb involved in case of the communication of an artery with a vein swells quickly from the flowing of arterial blood under pressure into the venous channels, and a vibratory thrill and murmur are found, the latter with systolic intensification. The cases may go on for years without extremely serious results.

## 11. DISEASES OF THE ARTERIES

### ARTERIOSCLEROSIS

This is a chronic thickening and degeneration of the coats of the arteries, leading to impairment of their elasticity and caliber, and compromising the functions of the organs supplied through the damaged vessels.

**Etiology.**—The causes of degeneration of the arteries are those which damage their structure directly, or those which lead to an increased burden which they must bear. In the former category we may place, firstly, the natural wear of life, for sooner or later, the arterial coats degenerate under the constant strain of use. Whether this degeneration begins in the earlier decades or after middle life depends upon the natural resistance of the tissues. Heredity is a factor of much importance here.

Next come those diseases and intoxications which damage the arterial coats. Syphilis is the most important one. I have seen death in the *eighth* year from the most advanced arteriosclerosis in a girl with hereditary syphilis. Typhoid fever is not infrequently followed by an early development of arterial degeneration. In chronic Bright's disease, after various infectious fevers, and in tuberculosis, arterial thickening is common. I have noted it frequently in the fourth decade in subjects of chronic digestive disease, especially in ulcer of the stomach, with pyloric involvement and consequent interference with nutrition.

Amongst other causes of arterial damage should be mentioned lead, long recognized as an important factor; alcohol, not at present regarded as of as much importance as formerly; tobacco, perhaps of more influence than heretofore believed, and especially in the causation of coronary sclerosis, with which angina pectoris is so often associated; tea and coffee, doubtless of importance if abused, but perhaps overrated by most authorities so far as they are customarily used; and finally the chronic intoxication of gouty disease. Adrenalin has been charged with the experimental production of arteriosclerosis in the laboratory. I have reported the case of a physician

who used over 200 ounces of the 1/1000 solution hypodermically for asthma, in the course of six years, without any evidences of arterial harm. Exposure to cyanid fumes in reduction works is a possible cause of early arterial sclerosis, but I know of no proof of its action.

In the second group should be placed most prominently the influences which create hypertension, notably severe muscular strain, mental strain, over eating and possibly adrenal over-activity. The mutual relationship between arteriosclerosis, renal disease and high arterial tension are well recognized, but cannot be profitably discussed here. In probably a majority of cases of early arteriosclerosis several factors are at work, the vessels being damaged by syphilis, alcohol, and tobacco upon the one hand, and the high tension of modern business life increasing the arterial burden, upon the other. In case of very early development of the disease, syphilis must never be overlooked.

**Pathology.**—The slightly raised yellowish patches in the aorta are typical of the condition, and are present in most of the vessels examined after middle life. Softening and degeneration of the deposit occurs, or calcification, either alone or in conjunction with the softening mentioned. The middle coat is primarily involved, a hyperplasia of the subendothelial connective tissue occurring as a compensatory process. The development of aortic aneurism is at rather an earlier age than that at which marked calcification of the aorta is found, signifying that the weakening of the coats giving rise to the former condition is perhaps due to the lack of the compensatory process mentioned.

In the diffuse form of arteriosclerosis the entire arterial tree may be involved. Hypertrophy of the muscular coats occurs, and the associated cardiac hypertrophy testifies to the increased vascular tension of the condition.

In the senile degenerative process the calcareous, tortuous, rigid condition of the vessels, and sclerosis of the aortic valves, are associated with the presence of calcareous plates in the aorta. In the radials, the nodular, pipe-stem type of thickening is extremely common.

In the mesaortitis of syphilis the spirochetes have recently been

demonstrated, giving confirmation to the belief that syphilis is the real cause of aneurism in at least a majority of cases.

In the pulmonary artery, under conditions of increased pulmonic tension, in the pulmonary veins, especially in mitral stenosis, in the portal veins in obstructive hepatic disease, and occasionally in the systemic veins, sclerosis may develop, with the same general pathology as we have noted in the case of the systemic arteries.

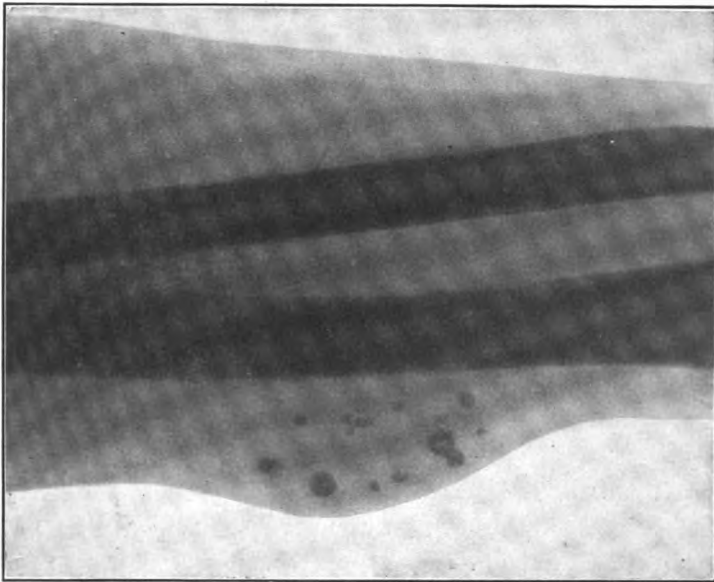


FIG. 45.—NUMEROUS PHLEBOLITHS IN VENOUS VARIX. (Photograph by Dr. G. H. Stover.)

**Symptoms.**—These may be general or local. Under the former heading we may first consider the symptoms of general disease. Although not constant, the increased arterial tension is the most characteristic feature. The rise in pressure may be from the normal 120 to 140 mm. up to 200, 250 or even 300 mm. In most of the cases of great increase renal involvement is found as well, and in almost any case a trace of albumin and granular casts are to be found, if repeated examinations be made. In the absence of definite renal disease we may still find increase in the quantity of urine, low

specific gravity and night micturition. The patient may complain of indigestion, frequently of inability to sleep well because of the feeling of oppression in the sternal region so commonly associated with the high tension, of dyspnea upon exertion, and more frequently still, of lack of bodily endurance. So striking are the two latter features that any patient with gray hair and an appearance of approaching senility is to be suspected of an arterial degenerative process, if they are mentioned.

Various local manifestations of the arterial disability become manifest. If an obliterative endarteritis affect the vessels of the extremities, as is so frequently the case in the foot and leg, senile or arteriosclerotic gangrene results. One of the most frequent sites of the vascular change is to be found in the coronary arteries. Working under more continuous stress than any others in the body, they often degenerate, while palpable evidence of thickening in other arteries is yet wanting. The various cardiac degenerations described elsewhere, are more or less intimately dependent upon this process, and angina pectoris is presumably directly dependent upon it. Marked dyspnea is frequently the most striking symptom of coronary sclerosis, and deserves more extended consideration than it often receives from the practitioner. As a result of the muscular degeneration we have dilatation of the heart, the development of relative leakage at different orifices, especially the mitral, and the general symptoms of cardiac exhaustion. The cases are to be distinguished from the ordinary ones of advanced valvular disease by the absence of early history pointing to the latter, and the presence of the usual features of beginning senility, even though the patient be in the fifth decade only. Coronary obstruction, angina pectoris, aneurism of the heart wall and sudden death from acute obstruction, rupture or other accident are further features of the affection.

In the brain there are two sets of symptoms dependent upon the vascular changes. The vessels are prone to spasm, producing often, sudden anemia, with failure of function of the portion of the brain supplied. Sudden dizziness, transient hemiplegia, or monoplegia, aphasia, convulsions or other manifestations of sudden shutting off of the normal blood supply are common. A hemiplegia may give all

the evidences of origin in cerebral hemorrhage, and yet clear up absolutely in a few hours. These are the vascular crises described by Pal.

The senile changes in the brain dependent upon the lack of sufficient blood supply, and manifested by forgetfulness, apathy, failure of mental power and eventual senile dementia, are very common after the fifth or sixth decades. The occurrence of cerebral hemorrhage depends upon arterial degeneration, commonly in association with increased vascular tension. Cerebral thrombosis is one of the commonest of clinical pictures, and depends upon the atheromatous changes of senility, unless syphilis or other conditions be present as a cause. In the vessels of the abdomen the changes described give rise to the so-called *angina abdominis*, resembling in its general manifestations the usual anginal attacks. Marked sclerosis of the superior mesenteric and other vessels is present in certain of these cases.

If the peripheral vessels be affected the patient may suffer from muscular cramps or more typically, from attacks of intermittent lameness. Upon the level such a patient may proceed comfortably at a slow pace, perhaps, but upon the attempt to move faster, or more especially, to climb stairs, severe cramps, and a feeling of painful exhaustion in the muscles

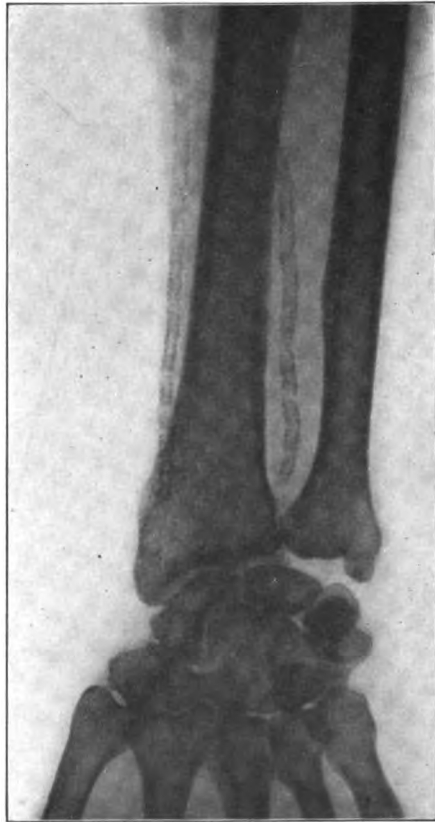


FIG. 46.—ATHEROMA OF RADIAL AND ULNAR ARTERIES. (Dr. G. H. Stover.)

compel a cessation of effort. The leg muscles are typically involved, and absence of pulsation in the dorsalis pedis artery is frequently noted. A similar involvement may be found in the arms or even in portions of the trunk. As in the case of angina pectoris, the over use of tobacco is believed by certain authorities to be of great etiological importance.

**Aortic Sclerosis.**—Aneurism has been discussed elsewhere. As a result of the atheromatous process in the aorta, dilatation results in certain cases. Those affected are especially males in middle or advancing age, often with a history of syphilis, or of one of the acute infectious diseases. The colored race is especially subject to the affection. The usual causes of arteriosclerosis are operative.

"The entire thoracic aorta may be so dilated as to give a wide shadow upon the X-ray plate.

"An atheromatous aorta causes a denser and more sharply outlined shadow than does the normal vessel." (Stover.)

Occasionally the aortic ring yields, and a relative regurgitant murmur is to be heard. More commonly an organic insufficiency exists as a result of the sclerotic changes about the aortic orifice. Tachycardia is a frequent symptom. Pulsation may be felt at the sternal notch, and is especially well marked in the descending aorta as viewed by the fluoroscope in certain cases. Pain, dyspnea, anginoid attacks and the features of circulatory failure are commonly noted. Increased area of mediastinal dulness is often appreciable. The "clink" of the aortic valvular closure is often well marked. The vascular tension is frequently less than the general features of the case would suggest. The radial pulses may be unequal, due to distortion of the emergent vessels at their origin from the aorta. Tracheal tugging may be present. The attacks of pain are stated by McCrea to be generally less severe than in true angina pectoris, and especially to affect both arms rather than the left one alone as in true angina. The immediate outlook is rather better than in aneurism, but the eventual prognosis is the same in both.

**Diagnosis.**—A history of any of the conditions described, with the signs of beginning senility, or with palpable hardening of the arteries, loss of pulsation in certain of them, commonly an increase

in the vascular tension, the urinary changes mentioned, hypertrophy of the left ventricle, aortic accentuation, and thickening of the retinal arteries should suffice. The increased blood pressure is one of the most important elements in the diagnosis. Without such increase many of the more serious results of the arterial disease may happily be wanting. In certain cases the Röntgen ray is of value in showing the condition of the arterial walls.

**Prognosis.**—The essential thing is an appreciation of the fact that arteriosclerosis means that the machine is wearing out. Not uncommonly the advance of the disease is a rapid one. Barring vascular accidents and angina pectoris the general outlook is that of gradually developing senility. In no condition does the patient accept the diagnosis and the prognosis of his disease with less grace than in the condition we have described.





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